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Human Costs of Electric Power Generation

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Human Costs of Electric Power Generation

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SYMPOSIUM ON ENERGY AND HUMAN HEALTH: HUMAN COSTS OF ELECTRIC POWER GENERATION

Sponsored by

Graduate School of Public Health
University of Pittsburgh
and
Ohio River Basin Energy Study

March 19-21, 1979
Pittsburgh, Pennsylvania

OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
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FOREWORD

The Ohio River Basin Energy Study is a multi-disciplinary project of considerable magnitude, supported by a grant from the Environmental Protection Agency. It is concerned with present and future energy - environmental relationships in the Ohio River Basin: a tremendous resource which, as Senator Bayh described it in his address, is shared by the people of six states; Illinois, Indiana, Kentucky, Ohio, West Virginia and Pennsylvania.

The Ohio Valley is the acknowledged center of coal production in the nation. Typically, five of the six valley states lead the nation in annual coal production. However well known its coal production is, the valley also has a unique nuclear history. The Shippingport Atomic Power Station, situated a few miles down the Ohio from its beginning at the confluence of the Monongahela and Allegheny Rivers in Pittsburgh, was the first commercial nuclear power station in the world. Shippingport began to produce electric power in 1957. The major thrust of the Ohio River Basin Energy Study (ORBES) is the evaluation of the many possible consequences of present and projected electric power generation in the valley. Its major objectives are to conduct an assessment of the environmental, economic, social and institutional impacts of all types of energy development along the Ohio River and within its basin.

A major concern of ORBES is the health component of energy resource extraction, transportation, conversion (i.e. burning of coal and reactor operation) and transmission. In order to establish a sound "state of knowledge" base it was decided to organize a three-day symposium devoted exclusively to the energy-health relationship. The six sessions were devoted to:

I) occupational problems in coal and uranium mining;
II) methodological problems in detecting health effects;
III) health aspects of fossil-fueled power plants;
IV) health aspects of transportation and transmission;
V) health problems in nuclear power generation; and
VI) future areas of concern.

The symposium was designed to provide ample time for discussion and the record of the proceedings, included in this document, attests to the areas of controversy and those deemed to require continuing investigation.

The future of electrical energy development in the Ohio Valley and beyond hinges to a considerable extent on the environmental and health consequences of its production, transmission, and utilization. We believe---and hope---that this symposium has provided a firmer foundation for the health assessment task of ORBES.

Dr. E.P. Radford

Prof. M.A. Shapiro
Graduate School of Public Health
University of Pittsburgh

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THE OHIO RIVER BASIN ENERGY STUDY (ORBES)

Birch Bayh

U.S. Senator, Indiana

Chancellor Posvar, Dr. Radford, Dr. Shapiro, Dr. Griffin, distinguished guests and participants in the symposium, I'm glad to be here. I thank Dick Caliguiri for his introduction, excessive as it was. I'm not too sure what the heredity of this ORBES Commission is. I guess I have to claim at least part of the bloodline.

I must confess to you I always think it's good for us to have the kind of experience that makes it possible for us to listen to that kind of introduction I got without inhaling. Such as this morning's plane ride from Washington, the young lady was going down the aisle taking our destination and name and she got to me and she asked the destination, and I said Pittsburgh, and gave my name. A big smile of recognition, "Oh, that's just like Birch Bayh, isn't it?"

You know, Mayor Caliguiri and I were talking about the environment which those of us as public officials serve. He has the toughest job in the body politic in my judgment. Being a Mayor he cannot afford the luxury of getting on a plane and going back to Washington this evening and I'm sure that the job has gotten even more difficult than it was at the time a certain occurrence transpired in the southern part of our state several summers ago, back when the trains were running reasonably on time and most of them were carrying passengers. A new minister arrived in a little southern Indiana town down close to the river in the jurisdiction of ORBES and he had been expected for a long period of time. In fact, they had about given up hope, so there was nobody there to meet him. He looked around and found no one and finally saw an old fellow sitting on the board seat of his donkey cart, waiting over under the shade of a big oak tree and asked the fellow if he'd carry him over to the parsonage. And the fellow did, but the poor old donkey just gave up the ghost and died right there on the parsonage lawn. The minister got down and offered his sympathy to the gentleman and went inside and immediately called the Mayor, "Mayor, this is Parson Brown, I just arrived in town." The Mayor interrupted and said, "Oh Parson, we've been waiting for you. I'm sorry I wasn't there. I had some very important business. Is there anything I can do to help you?" "Well, Mayor, you can. There is a dead donkey on my front yard and I'd appreciate if you take care of the remains." "Well," said the Mayor, "I'll take care of that right away."

Well this is in the middle of the summertime down southern Indiana and on the morning of the third day some of that pure unpolluted southern Indiana air came into the bedroom of our minister friend and it came to his attention that the Mayor had forgotten his promise. So he bounced out of bed, whipped over to the telephone and dialed the Mayor and said, "Mayor, this is Parson Brown. Three days ago you told me you were going to remove the remains of that dead donkey. It is still there. What's the matter, don't you politicians keep your promises?" The Mayor answered rather hastily, he had been up the night before dealing with some of the problems with his constitutents and he said, "Well, Parson, I thought it was the duty of the clergy to bury the dead." The Minister thought and said, "Perhaps you're right, Mayor. I was just advising the next of kin."

Mayor, I think you and I and any of us in the body politic and the political hierarchy are living in a time when it is difficult to get the benefit of the doubt. I must say the kind of sensitivity you have shown, first as a council member, and now as a Mayor of this fine city is an example I am sure others would like to be able to follow.

I am glad to have a chance to be here, to share some thoughts with you about the problems of environment and energy. The reports that have reached me, perhaps not totally accurately, but I think fairly accurately, about the environment and atmosphere of the meeting this morning, makes me wonder if I'm not like the early day Christian who was thrown into the Colosseum and as he awaited the lion he got down on his hands and knees and bowed his head and began to pray. After what seemed like an endless period of time nothing happened and he looked around and there the lion also was kneeling in prayer. The Christian threw his head to the sky and said, "Thank you, Father, for delivering me." Whereupon the lion opened one eye and said, "Shut up. I don't know what you're doing but I'm saying grace."

Perhaps some of you will agree and perhaps some will disagree but I think the spirit of friendly controversy, the spirit of prodding and the spirit of really searching for the real answers to the problems of energy and environment, that is the only kind of attitude, in my judgment, that is going to help us solve both of these problems. Now what I would like to do, this noon, is to perhaps go through some notes that I have written down in consultation with my staff to try to sort of put down a foundation, particularly for some of you who do not live in the immediate valley area, to describe the way we got here as far as the establishment of ORBES is concerned. I'd like to discuss the origin of ORBES and, in general terms, the "politics of policy." And then open for more specific questions. The latter part will be the most beneficial for both of us.

I am grateful for this opportunity to speak before this distinguished group brought together by Dr. Radford under the auspices of the Ohio River Basin Energy Study, which most of us who are at all familiar with this know that that is ORBES. We appreciate the fact that we're here but I think Dr. Radford, as important a role that you played, I think the facts are that neither you nor ORBES has actually brought us together, but it was an act of nature known as the Ohio River, the nature of which is very, very precious to all of us up and down this river. It was that act that really brought us here, resulted in this common resource shared by the people of Indiana, Southwestern Pennsylvania, Ohio, West Virginia, Kentucky, and Illinois, and there is no questioning the fact that it is an extremely valuable asset.

But to share the river's assets is to also share her problems. And her problems are not unique to this region. Other places in the country may be represented here. I thought that perhaps discussing how we got to the place of organization where we are now, studying this problem with the regional approach, that might be helpful as you apply this to other places. We have all sorts of common strengths and weaknesses here.

I recall some of the distillery workers in Lawrenceburg, Indiana, having some friendly repartee with some of their workers who live across the line in Hamilton County, Ohio, in Cincinnati, and which starts out usually where Cincinnati people say: "Ah-ha, to tell you what we think of your folks in Lawrenceburg, we flush our toilets and dump our sewage in the river and it goes down for you to drink it." And the people in Lawrenceburg say: "Well, it's not quite that way. What we do is bottle it up and sell it back to you." I'm not too sure that settles the problem but we are inextricably bound together and for that reason we have tried to approach this in a common study vein.

I happen to believe that the energy-health dilemma, faced by those of us in the Ohio River Valley, is indeed, if it isn't already confronting, will soon confront most other areas of America. ORBES, though regional in nature, is not provincial in scope. We are facing a national challenge of unprecedented complexity and I am proud of the fact that we in Ohio Valley have taken the lead in developing new strategies to face this challenge. I am extremely proud of the work that is being done by ORBES and hope that we can conclude it as rapidly as possible, and then be able to implement some of the findings.

If we look at the real historical foundation, on which ORBES is based, it really didn't start here in the United States. It started back in October, 1973, when a group of non-industrialized countries in the Middle-East arrived at an agreement to reduce the flow of crude oil to the United States, Europe and Japan. The Arab oil embargo plus OPEC traumatized this country because it struck two simultaneous blows from which we are still reeling. And some of us are asking ourselves: is this really true? One of the blows was political. The American public became aware for the first time, during the embargo, that the country was now vulnerable in a vital matter, its energy supply, really its economic jugular vein.

The other immediate impact was economic. Across the nation the higher prices of imported and domestic petroleum worked their way through the chain of economic interdependencies until they appeared in the form of increased prices for other kinds of energy and energy intensive goods and services. This economic blow; this incredible inflation attendant to the high price of energy which still dogs us politically and economically today. The subject of OPEC, the merit or the fallacy of a so-called free market for energy, the discussion of those issues it seems to me can better take place at another time. But, I think it is important to start out with the understanding that this is really the first time in the entire history of this country that we really began to worry about the cost of energy and the availability of energy and what that can do to the entire economic structure of this society and the way of life as we have come to know it. It is the first time we have begun to assess investments, lifestyles, in terms of their impact, and the impact of energy on them. When you take this sudden awakening to the costs and availability of energy and you lay it on the foundation of a decade of concern and progress dealing with the environmental problems that had gone long unattended in the society, you bring us to the foundation, really the two pedestals, on which the ORBES study was based.

A national consensus emerged during that period in which we were trying to come to grips with reality that an energy supply was threatened. A consensus emerged that we must reduce our reliance upon foreign oil and place greater dependence on domestic fuels. In the electric power industry there was a strong push that we must have a greater reliance on nuclear energy and we must site more new coal fired plants near the coal fields. I must say the closeness of coal is not the sole criterion for plant sites as far as the utilities were concerned. Water for transportation and cooling, as well as the existence of these plants in reasonably sparsely populated areas, were also required as people looked at the criteria of development. Portions of Indiana and Kentucky along the Ohio River between Cincinnati and Louisville apparently met these specifications.

So we found the Arabs hardly lifted the embargo when several utilities announced their intention to construct many plants on both the Indiana and Kentucky sides of this reach of the river. It was significant that certain of these plants were long distances from the consumer-load centers of the utility. In other words, we found unusually long spaces, great distances existing, between where the utilities are going to produce much of this electricity and where it was ultimately going to be consumed. Thus, it was clear that external conditions were encouraging power plant construction in a scenic stretch of the valley and that much of the power would be exported while local residents were saddled with the liability of potential environmental problems. It is not quite that simple, as some of you know who have looked at the early results of the ORBES study and who are familiar with the air currents, that indeed many of the environmental impacts are going to follow, if not exactly, at least a general direction of much of the use of the electicity and it is that kind of problem we're trying to dissect and have a better understanding of.

Citizens representing several environmental and public interest groups called upon me early in the fall of 1974, to report the problem and that more importantly they were unable to get answers from the various government agencies on the impact of these plants. Likewise, they were unable to find out just how serious the environmental problems might be from such a heavy concentration of facilities. Nobody seemed to have the answer and so they asked for help. Thus the Senate Appropriations Committee, of which I happen to be a member, did act to direct the U.S. Environmental Protection Agency to conduct a far-reaching study of energy development, including power plant construction, in the lower Ohio River Basin. The committee's action took the form of language which was added to the report in the 1976 fiscal year funding measure for EPA and the language added in July 1975 reads as follows, just to give us a little idea to remind ourselves just exactly what the legislative mandate for the Ohio River Basin Study was and is.

> The committee is aware of plans in various stages of development which could lead to a concentration of power plants along the Ohio River, in Ohio, Kentucky, Indiana, and Illinois. Although the environmental impact of such a concentration could be critical, the decision making authority regarding the construction of these facilities is dispersed throughout the federal government and state governments. The committee directs the Environmental Protection Agency to conduct, from funds appropriated in this account, an assessment of the potential environmental, social and economic impacts of the proposed concentrations of power plants in the lower Ohio River Basin. This study should be comprehensive in, scope- investigating the impact of air, water, and solid residues on the natural environment and residence of the

> region. The study should also take into account the availability of coal and other energy sources in the region...

When the Appropriations Committee first headed the mandate to EPA we were concerned about the concentration of plants in the four states cited in the report: Illinois, Kentucky, Ohio and Indiana. A number of factors, including early findings which indicated that plants' air emissions were carried hundreds of miles, led us to encourage EPA to expand the ORBES region to include Southwestern Pennsylvania and most of West Virginia. Thus, all six states, which border the Ohio River are now included in the official areas of the ORBES study.

Both EPA and the Appropriations Committee gave much thought to the institutional structure which might best carry out the energy assessment. I think we arrived at a rare combination here. We encouraged EPA to consider a framework which would include representatives from University faculties in the Ohio River Valley. In an innovative manner EPA sought proposals from major universities on how they might cooperate in the undertakings, and the ORBES study is the result. Most of you know, researchers from nine university campuses in these six states are engaged in a multidisciplinary endeavor.

I might add that one of the bonuses of expanding the ORBES region was the opportunity to involve representatives from the University of Pittsburgh's distinguished School of Public Health. In the second and third years of this activity we were grateful for that opportunity. Of course, Professors Radford and Shapiro of the School of Public Health are now participating in ORBES and have taken the initiative in convening this symposium. Their examination of the public health consideration is one of ORBES' most important areas of inquiry, and I look forward to studying their findings as quickly as we possibly can.

From the beginning, those of us in the Congress realized that the assessment would have little credibility unless the University researchers were given independence from political and bureaucratic interference. Thus researchers on an ORBES core team are participating in the project under separate grants awarded directly to their own universities by EPA. An EPA Project Officer, frankly an internationally respected environmental scientist, Lowell Smith, coordinates, as we know, the nine university activities in an effort to guarantee that funds are being administered in a manner consistent with federal guidelines. But ORBES researchers are accorded freedom of inquiry in all respects. In addition to the ORBES core team, more than one hundred other faculty and staff members in the various universities are providing support research to strengthen the undertakings. Realistically, of course, there must be coordination, guidance and direction in such a large project. Such direction would be suspect if it came directly from EPA itself. As we know, EPA is clearly not, and frankly should not be a disinterested party.

To provide such direction in the university context, EPA awarded grants to two University of Illinois members of the core team to serve as a management team for the total ORBES effort. I believe that the selections of Dr. Stuckel, a distinguished engineer and Dr. Keenan with his experience in political science, were outstanding. Their task is, of course, to assure that the research process in preparation of the final report be as objective as possible. EPA and Congress have placed a heavy burden upon both the management and research elements of the ORBES endeavor, one that may be too heavy, according to some prophets of doom. Indeed, they look at the past, and they say that in most instances the record of inter-university cooperation and research has not been overly impressive. Some say it is a contradiction in terms for a federal agency and such a large number of universities to attempt to cooperate in such a fashion. After all, the main function of a federal agency is to keep the wheels of government moving while a primary purpose of university research is to criticize that movement. Well, frankly, there may be some truth in this at times. I think we should look at the positive side of this marriage which I think can bring tremendous strengths to bear and I am not willing to accept the conclusion as inevitable from those who doubt whether we can succeed. Neither am I willing to accept as inevitable that, because EPA is coordinating ORBES, the agency is seeking only those findings that conform with conventional EPA wisdom. I know well that EPA has been criticized for such a policy in the past. But, I am confident that this will not be the case for ORBES.

I think it is important for us to see that the ORBES study and those who are out looking for the answers and putting together the data on which conclusions will be based; that they be immune from political pressures. I think so far we have been successful. We should take the politics off finding out what the facts are. I have to say that I think we have to distinguish that very important goal on one side, from the very realistic assessment of how the real world operates on the other. And that is that political decisions are the way public policy is made in America today and to expect that the so-called politicians, the elected public officials, will have no opportunity to weigh the factors involved is to expect something that cannot and, in fact, should not exist in our society. You may want to get into a discussion of this in a little more detail because I understand there may be some differing opinions and that's what makes the world go around.

Now, I think it's important for us to have the benefit of all this information as quickly as we possibly can. The economics of environmental regulation and economics is one of ORBES' major areas of inquiry, as we know has become a very hot topic. Those of you who just came from the mining seminar are undoubtedly aware of the recent fracas that ensued in Washington when certain of the President's economists were critical of the proposed surface mining regulations on the basis that they were too costly.

For those in Congress, and I should say what I'm about to say without relating that specifically to the assessment that was made in Washington by some of the President's economists, but looking at it more generally, there are those in Congress and the agencies, probably some of you in this room, who believe that money should not be a factor in determining our national environmental quality goal. Let me say as one of the original sponsors and continual protectors of EPA, and the goals it was designed and is still designed to accomplish, that I must say nevertheless that I disagree with this point of view. I believe that environmental decision makers, those in Congress, the agencies, the research institutions, must be able, with some degree of certainty, to have the sense of the cost associated with their decisions. If I am to make any contribution to a symposium such as this, I suppose, and I scratch my head to see what kind of contribution I could make, in the presence of those who are experts in the science of both energy and environment, it seems to me if there is any contribution that I as a United States Senator could make, it is to share my very frank assessments, complete with their strengths and their weaknesses, of the practical aspects of policy implementation. And to do this in the general fashion and then we can get into some of the specifics in the question and answer part of the program this noon. Now, I confess that I probably have already stepped on some toes and am about to step on some more. I don't intend to apologize for that; I found that in most of my lifetime in dealing with public policy that probably we need a good deal more toe stepping, because then you increase the circulation and you get a little more action.

In both this area of energy policy, and environmental policy and particularly the coordination of the two together, we are woefully short of action. If I may just share some thoughts of my conscience with you: from a moral sense I am not prepared to trade dollars or jobs for lives. I find it very difficult to do that, impossible to do. However, I am equally concerned over the fact that we've now become polarized; those of us who were early on in the movement trying to prick the national consciousness as far as the environmental degradation that is going on, we are polarized more and more from those in society who say we must have more jobs, period. For those on the other side who say no, the health question is the only question we must consider. And it just seems that together we had to find ways to stop this polarization because the real test of our free society is how we can have enough jobs to accommodate a growing society, at the same time we recognize the important public responsibility of having a legislative policy that will protect the health, not only of this generation but of the next generation of Americans.

It is easy to say that, I have to confess to you. It is much more difficult to implement that kind of policy and to get the tradeoffs at an acceptable place on the scale. It is complicated by the fact that the loss of jobs, the cost of environmental protection, are immediate and obvious. The loss of lives, more particularly the deterioration of lives, which will be felt ten or twenty years from now, is not immediately weighable. You can look at the admissions to our hospitals of those who have respiratory ailments, and you can chart the almost identical line with the particulate matter in the air. But it is not the kind of fact that is felt by those who are alive and working in a healthy way in the workplace today. That is why I think it is extremely important for us to look at the health factors and look at the economic factors and do a better job of trying to mix both of those together at an acceptable level.

I think we have to do a better job of articulating the health realities, not just the immediate cost of environment as they damage health, but the long range relationship of environment to health. Secondly, I think we need to have a better assessment of economic consequence, real economic consequence--how they play on health, what dimensions the health laws will really be. If you're going to really be able to weigh this off we have to have all those facts so we can make intelligent decisions. I think it is possible to do that and ORBES is going to be very helpful to us. But this is a critical kind of decision that we just have to be prepared to make.

We in Congress have to take better advantage of our research arms such as the Congressional Budget Office, the Office of Technology Assessment, to be able to assist us in fixing a price tag to the environmental goals we legislate. And as controversial as it is, there may be a point at which a standard that makes one microgram increase in the requirement, makes it that much more stringent, might be incredibly costly without any real returns from the standpoint of public health. I think we have to make those decisions, and hopefully to make them accurately. I have to say this from a practical standpoint, at a time when we are not able to prove, as I think we can if we do it the right way, prove the real health benefits that are necessitated as a result of certain environmental requirements then the political, in the proper use of that word, the political support for environmental protection is going to be gone and the whole effort will be lost. So it is important for us to have all these facts and be able to do a good job of selling. This is true not only of Congress but the regulatory agencies.

Another matter of some controversy, I think, causes me to say to those of you who may think contrariwise that design specifications often reduce the ability of the marketplace to come up with alternative and more economical ways to handle the waste products of our industrialized society. I ask myself, "Isn't it far more prudent to set a standard and allow the polluter to reach it within a reasonable period, using whatever technology makes sense for his individual enterprise?". I'm not so wed to the vehicle, the technology, as I am to the results. And I think we need to have enough flexibility to let each situation be resolved on its own circumstance.

I have to say that as I've been personally involved in this issue with some of my communities and some of the interest groups that I represent that they are characteristic of America. I find that the most frustrating realism in public life today is to see that there tends to be major conflicts, just irreconcilable conflicts, between groups that really benefit or lose from the same set of circumstances. You get industry on one hand and consumers on the other, you get the auto industry, you get government at loggerheads with one another. Somehow or other we have not been able to get everybody together, and say okay, it isn't good for industry to let the environment degrade to the place that you have the kind of major movement that is not going to consider any of the economics at all. It isn't good for them, it isn't good for the automobile companies, to have the kind of situation we had with the Clear Air Act which I was and continued to be a strong supporter of that legislation. I have to confess to you that I don't think any of us in 1970, when we sat there in all those hearings thought we were really going to get an absolute decision that utopia is going to arrive in 1975. By the same token, the people in the auto industry that had all the technology to tell us to stop polluting, as we burn hydrocarbons in internal combustion engines, they weren't willing to share that information at all. They said, "We can't do it.", period.

So we had to do something to get their attention. So we established an arbitrary goal and said, "You better do it by 1975." Well, industry rushed out here and instead of government and industry and consumers and everybody sitting down and saying, "Okay, here is the goal we're going to reach. How is the best way to reach it?" You had industry on one side, saying, "We can't reach it.", and government on the other saying "You're going to reach it by 1975." We ended up with a catalytic converter, which I think most of us understand is certainly not the best long-range solution.

You know it doesn't help the purpose of our symposium for me to pour out some of the frustrations in my heart as far as I look at this as a policy maker. But some of you who are providing these facts, I'd like for you to know it is not always an easy solution as to how you get these groups together to try to put together a policy that is really in the long term national interest of the entire society. You look at the ambient air quality problems that we have right now, in which some of our communities are going to be confronted in a relatively short period of time, with major sanctions if they do not meet the standards that have been established. I think in some communities this is going to cause significant dislocations if they're not able to reconcile the pollution with the standards. I have been trying to deal with this and I must say as frustrating as the first example is I have been heartened by the second. If you live in Terre Haute, Indiana, you're not as concerned at all with what the best way to solve the problem in Buffalo or Detroit is, you're concerned with how you solve it in Terre Haute, Indiana, or Indianapolis. Yet we had, for a long period of time, EPA sitting there absolutely unwilling to have any adjustments that would take into consideration local situations. Well, we sat down with EPA and we sat down with some of our institutions of higher learning that had the technology and I must say I've seen an amazing willingness on the part of EPA to say, "All right, we're not going to say you have to do it our way as long as you recognize the need to do it and are willing to commit to reaching that goal." On the other hand, you have some in industry who have been totally unwilling to recognize the fact that they're even polluting at all say, "All right, if you let us have the chance to put this together in a sensible way, we're going to sign off and we'll meet certain standards." Now, I have to confess to you that the jury is still out on that. Some of those people who had been the major polluters may be using this as a guise, I don't know, to put it off another year or two or three or four, five years. I hope that is not the case. Although I am willing to have a little give and take on how we reach a given goal as long as we reach it, I'm not willing to let any of those who are really destroying our environment and significantly diminishing the lifespan of many of our citizens, hide under a cloak of reasonableness to avoid what is their ultimate responsibility.

Forgive me for sharing some of those personal experiences with you but I think what we're trying to do in ORBES is to get that information out there as quickly as we can. I would hope that writing of the final report will commence in January 1980. That is really just around the corner considering the size of the job. I hope we can get that out there as quickly as we can. Give us a set of options; let us weigh the pluses and minuses and the strengths and the weaknesses of these given options, and I hope that we in the Congress, once we have the wisdom that will come from knowing all the factors involved in the development of energy and its impact on the environment of this region, once we have the wisdom, I hope the good Lord gives us the courage to do what is right. Right for the health of our communities, really a significant section of our country. At the same time we realized the importance of providing sufficient energy for many other parts of the country to meet the standards of living that they have become accustomed to.

I may have had some pessimistic notes slipped in here. I am normally an optimist, I still am an optimist. But I think that optimism has to be laid on a realistic scale and I am confident we can meet the dual goal of a good environment and an economic plane that is acceptable to our society. We cannot reach these two goals if we try to kid ourselves, if we're not realistic. The costs involve both failure to provide good environment and the cost of providing good environment. We have to know both.

Now that is the end of this filibuster and this rambling around. What's on your mind is of more significance than what I've talked about.

Discussion

Dr. H. Spencer, University of Louisville: I am one of the ORBES core team members, also one of the citizens that knocked on your door. We're grateful to you, Senator. I have long said, and like to say here, that in those early years we had more access to your office than we had to the EPA.

Senator Bayh: I'd be glad to claim them; I just don't want to claim all the credit. I think you fellows really were the fathers. I was just offering the environment in which you could do your work.

Dr. H. Spencer: I have one question which I think you could give some answer to. There are a number of industrialized countries that have no energy resources and yet are economically strong. Those countries import virtually all of their oil. We don't have quite that much of a problem. Which means they must be doing something right and we're doing it wrong. Answering those questions, being able to get at that, is part of the ORBES charge. What do you think we're doing wrong in the economic market as compared to these nations?

Senator Bayh: May I just offer a little advice? Ask another question. We haven't known each other long enough, and we have a friendly repartee. The reason I say that is that if ORBES tries, what are the consequences of what happens out there? I think if we get ORBES into that area you're getting it into an area from which you're never going to be able to extricate it and we should stick to some of the more immediate problems.

One of the most current problems is what's going on at the multilateral trade negotiations. I am a trade man. Looking at the value of the dollar--what that has done-- I have to say that I hope we can balance our budget. I think there has been far too much emphasis and far too much hope placed on that because that is one of the parts of the problem. But, if we look at the real problem of the deficit balance, the real problem of the payments and you look at the way our workers in our industry are treated one way when we try to trade on someone else's turf and they're treated another way when they try to trade on our turf. We have to come to grips with that, because that is basic as far as the strength of the dollar is concerned.

Here again I've asked myself the same question. The other countries are not doing as much of this as I think probably we are right now. One of the things I have deeply regretted that we have not really been able to get geared up, and we have started now and it's been woefully late in coming, is a major effort to try to provide alternative fuels so we could get this oil monkey off our back. That has a very insidious effect on us, not only from the standpoint of energy, but also politics, national blackmail. There are some things like that we just have to do more of. I think ORBES lies in a more confined context and I recognize that your question is right on target as far as the complexity of the overall problem, the ORBES findings is only a small sliver thereof.

Dr. Spencer: The health costs have escalated so terribly in this country, and since we are debating health here today which is such an integral part of the ORBES project, I feel we cannot really dissociate ourselves that easily from the subject. I know that you are not suggesting that. I'm trying to reemphasize the complexity of the health area in relation to the economic area and to the world energy problem.

Senator Bayh: You're right. One other problem we have is that we have demanded, I think rightly so, a higher standard of living in the environmental sense, than any other nation on the face of the earth. That is part of the problem. That's one of the benefits of our society but there are very few, if any, other nations, very few of any other taxpayers, that have to bear the costs of quality of life as far as environment is concerned in the production process as we do here in our society. The irony of this is if we look at a hundred years from now the global impact of some of the environmental practices that we're pursuing or not pursuing, this is going to come home to haunt everyone. And to the extent we can do more to try to prick the conscience of the industrialized world, all the industrial communities of the world, that pollution of this society is a world problem, not just an Ohio River Valley problem, or a United States problem, then it seems to me we recognize the environmental problem that is going to be confronting us a couple or three generations away. We also recognize some of the attendant economic problems that are a result of the United States awareness of this problem before other nations. I don't necessarily want to pass the buck to any nation but I think if you look at what we're doing in our society to try and deal with the environmental questions, it's a generation or two ahead of where the other societies are in most industrialized societies.

Question: Senator, you said that we have to have a better handle than we now have on the cost of achieving the environmental standard we have set for ourselves. We have certain very significant environmental standards imposed upon us by the Clean Air Act. And the Clean Air Act says nothing about how one can assess the cost to society of achieving those standards. Would you favor an amendment of the Clean Air Act, to impose that specific requirement upon EPA?

Senator Bayh: Whether it takes an Amendment to the Clean Air Act, or recognition of the real world in which the Clean Air Act is being implemented, I am concerned about the problem that once you start amending that Act you then open it to those who are not really looking for realistic kinds of weighoffs and tradeoffs, but many people who are looking for loopholes so that they don't have to meet any standards at all. I may be rather hard here. But here again I find it very difficult on one hand, to say that we weigh dollars against human lives. But in the long run, if we're talking about a de minimis, or questionable increase and health quality that hasn't been proven, hasn't been studied, versus an economic dislocation that is rather quick and apparent, then I think that it is an easy kind of a tradeoff to make. Unfortunately, many of the decisions that have to be made are not that easily recognizable.

Dr. Leonard Hamilton, Brookhaven National Laboratory: I recognize that many socio-economic variables are very important. The thing I do not understand is why environmental controls necessarily mean a depreciation of jobs or lack of jobs. The results suggest that they may create more jobs.

Senator Bayh: There are some studies that do show that, if you take the society generally, the studies that I've seen as an environmentalist, and I like to believe those studies, show that there are more people working now to produce environmental protection equipment than have been displaced by environmental requirements. I think we all understand that many of those people are working in areas where dislocation is not going to occur by the imposition of the environmental standards. The difficulty is that you have core areas of major dislocation. We try to go back and see where could we have done better and if we don't learn by our mistakes, particularly when we are pioneering in this whole area of environment in the United States. I think we have to get consideration of this now. ORBES is probably as good an example as any where you have major pollution problems created in one area to provide energy supplies to others, but that pollution is not going to remain stable so it is going to follow normal trends. I think we have to look at a broader base of financing our environmental requirements. I think we might have been better off if we would have permitted a significantly higher tax writeoff to get these things on stream quickly, so you don't have haggling that we're going to fight forever. Let's find a way in which we can finance this situation out there generally. The people that are going to be producing the energy and have the environmental protection equipment established, say in Madison, Indiana, since that is going to benefit society generally to get that electricity. Maybe society ought to generally help bear some of that and a tax writeoff would help to do that. With what has happened to utility rates, we see that the fact of life is that consumers are going to pay for one end of the scale or the other. And yet we are not being able, the way we are doing it now, to get the systems implemented as quickly as we otherwise could.

To get back to what we ought to be doing. We ought to be doing much more than we have been doing in research as far as combustion is concerned. Most of our research has been directed at energy supplies, not the more efficient or more environmentally sound combustion processes. We had two or three of them, the fluidized bed combustion process, just to name one, that is very close on, that can do a tremendous job both as far as SO2 and increased BTUs. Yet we have not emphasized that part of the equation. Now I could have answered your question in one word but a Senator never does that when a five minute answer is there.

We are prepared to look at where the regulations have us compared to what the original legislative intent was. But I think we would be wrong if we looked at that from the standpoint of retreat. Most of the goals that we originally established were reasonable and we didn't have nearly as much idea about how to get there, the cost attendant, the time and dislocation involved in some instances as we do now. So I think it is only fair and only wise to periodically have oversight and look and see where these regulations go. EPA is not the only government agency to put out regulations. And as Dr. Bingham at OSHA will tell you she couldn't see why her predecessor dealt with the size of outdoor toilets and the reasonable proximity of (don't get me started) wooden ladders and fire extinguishers and things like that. I mean there is something about regulators that tend to get carried away with dots and tiddles instead of great announcements of public policy.

So I think it is important to reassess where we are. But here again I would hope that we could find some way in which we could get these near irreconcilable forces together somehow. To get back to that auto thing, how you could have gotten somebody to get the auto people that had the technology and said, "Okay, we're going to make a major innovation in this internal combustion engine and we're not going to say never.", and we're going to say "Okay, we'll give you another four or five years." We're going to end up giving you four or five years anyway and we're going to have a mousetrap instead of a major change. So it is with some of the other air pollution problems.

May I just make one more comment? I would hope that those of you who are here and who obviously have deep concern and interest as well as more than casual expertise in this area, would let me know your assessment of this problem. It is not an exact science and I'm sure that those that are involved in ORBES are painfully aware that there are going to be some tradeoffs that are not exactly certain. And it is important for us to have the experience of benefit of as many different aspects of this problem as we possibly can, so we can make the right determination. As I say, you can provide us the wisdom, I hope the good Lord provides us the courage, because it is going to take a good dose of both of those.

Dr. Edward Radford, University of Pittsburgh: Thank you very much, Senator Bayh, and we appreciate your taking the time to come to the symposium.

SESSION I: HEALTH PROBLEMS IN EXTRACTION OF FUEL

Monday morning, March 19, 1979

Moderator: Edward P. Radford, M.D.
Professor of Environmental Epidemiology
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University of Pittsburgh

OCCUPATIONAL AND ENVIRONMENTAL HEALTH PROBLEMS IN COAL MINING
By
Curtis Seltzer, Ph.D.

RESPIRATORY PROBLEMS IN COAL MINERS
By
W.K.C. Morgan, M.D.

URANIUM MINING - OCCUPATIONAL AND OTHER HEALTH PROBLEMS
By
Joseph K. Wagoner, S.D. Hyg.

OCCUPATIONAL AND ENVIRONMENTAL HEALTH PROBLEMS IN COAL MINING

By

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Coal-fired electricity is not made cheaply. The work-related health and safety hazards of coal mining are well-known to miners and operators alike. Even the general public has come to appreciate the costs of "black lung" disease and accidents that coal miners experience. Many of the environmental health costs of coal mining are less familiar although coalfields residents know them well. These concerns range from acid mine drainage and fugitive dust to accidents from coal-hauling trains and flooding from stream siltation in Appalachian watersheds caused by years of contour strip mining.

Most major environmental health problems have been identified and a sense of their magnitude, severity and remedies are known. Current debate focuses on the level of magnitude, degree of severity and applicability of certain solutions. In policy terms these issues translate into a few simple questions: 1) Are current federal occupational and environmental standards adequate?; 2) Are these standards being complied with on a daily basis?; 3) Are the available compliance data reasonably reliable?; 4) Are current monitoring strategies adequate?; and 5) Are existing enforcement programs working well?

While most analyses discuss these questions and issues individually, coalfield residents experience them holistically, cumulatively and, in a sense, synergistically. Social turbulence in the coalfields is likely to be related to the set of adverse environmental health conditions miners experience in the course of their everyday lives. In these matters, scientific precision is often superseded by perceptions of reality. If the creek in your backyard is the color of orange soda pop, whether its pH is 3 or 6 is of little consequence.

Both the problems of and solutions to occupational and environmental health in coal mining are related to supply-and-demand economics. Tight markets and static prices determine the level of externalized occupational and environmental costs. When demand was sluggish, concern for environmental health was deemphasized as workers, environmentalists and community activists feared that economic pressure on local mine operators would affect their competitiveness and jeopardize jobs and community welfare. When demand is rising steadily or when prices are increasing (whether or not production is increasing), coal workers and community activists are likely to push coal operators harder for preventive and compensatory programs related to occupational and environmental health. As coal demand and production rises over the next 25 years, economic conditions will be created for increased demands for occupational and environmental safeguards.

To understand the nature of coal mining's current occupational and environmental health problems, it is necessary to understand something of the economics of the industry. Neither occupational health and safety nor environmental quality exist independently of coal's economics and politics.

The single most important economic fact about the American coal industry is that for most of this century coal demand was stagnant. The industry's capacity to produce did not change significantly after

1918. In fact, as recently as 1975, coal operators produced less bituminous and anthracite coal than they had in 1929; 658 and 655 million tons respectively. In 1947, about the same amount of coal was produced as in 1977.

Lack of demand, low prices and stiff competition forced coal operators to cut production costs as much as possible to survive in the 1920-1970 era. During the first 30 years of this period, the operators maintained competitiveness by holding down their labor costs despite wage pressures by the United Mine Workers of America. Labor costs were reduced by gradually introducing new labor-reducing technologies such as mechanized cutting, drilling and loading machines. In 1920, 640,000 miners were employed compared with 416,000 in 1950, a 35% reduction. After World War II, the industry mechanized rapidly and comprehensively. The introduction of continuous-miner technology, electric shuttle cars and roof bolts in underground mines after 1950 enabled the industry to maintain tonnage levels with even fewer miners. A section equipped with one continuous-mining machine could produce as much coal with 10 workers as a hand-loading section with 80. Labor costs were also reduced by the steady increase in the share of coal output mined by surface methods, which were inherently more efficient and less costly than underground techniques. The portion of total bituminous production mined by surface methods rose from 24% in 1950 to about 60% today. Underground mechanization and surface mining cut deeply into the workforce. The 416,000 bituminous coal miners of 1950 were reduced to about 125,000 in 1969, a cutback of 70%. Bituminous production, however, actually increased from 516 million tons in 1950 to 561 million tons in 1969.

This set of economic and technological changes carried grave implications for occupational health, safety and environmental health in coal mining. Weak coal demand and cost-cutting pressure meant operators minimized health and safety expenditures. Occupational health and safety costs were often externalized in whole or in part. (An externalized cost of production is not paid by the mine operator. It is shifted in one form or another to employees--as accidents or disease--the local environment, or adjacent communities.) The frequency of mine fatalities showed no improvement between the early 1950s and 1970,although the number of fatalities fell as the workforce was reduced. Similarly, the frequency of disabling injuries showed no improvement, although the number of injuries fell. In terms of occupational health, continuous miners increased the levels of respirable mine dust, which, caused many thousands of cases of respiratory disease to appear by the late 1960s. No Federal dust standards were established until the implementation of the 1969 Coal Federal Coal Mine Health and Safety Act in 1970. In addition, the environmental costs of surface mining were largely externalized in the 1950s and 1960s. A few states enacted environmental controls in these years, but they were inherently weak and enforced weakly. The environmental damage from unfettered contour surface mining was considerable--naked high walls run for 10,000 miles throughout Appalachia; overburden was pushed down hundreds of slopes without regard for landslides, stream siltation or water quality; blasting disturbed ground water and its noise aggravated nearby residents; fugitive dust from mining and haulage nettled many; finally, surface owners often had little say about how a coal company went about extracting the sub-surface coal it owned.

The economic conditions forcing coal companies to externalize occupational and environmental costs were also handicapping the capacity of coalfield communities to bear them. Hundreds of thousands of unemployed union miners and their families were cut off from the health care and retirement benefits they had earned. (Those union miners who were fortunate enough to continue working enjoyed first-dollar coverage through the UMWA-negotiated Welfare and Retirement Fund.) Federal "commodities" (surplus foodstuffs) became the dietary staples for many.

Most significant in terms of environmental health was the short changing of local tax systems, which prevented communities from building an adequate infrastructure of public services and facilities. Local communities, counties and states had rarely taxed coal production or undeveloped minerals sufficiently. They feared that doing so would disadvantage local coal in competition with non-taxed coal from other areas. The political influence of the coal industry was sufficient to beat back efforts to impose higher local property taxes or state severance taxes. The result was that coalfield communities lacked many facilities--such as water and sewage treatment plants, recreation and adequate housing--and services that make for community public health. The health consequences of this syndrome were documented in a 1947 report on coalfield health conditions prepared by Admiral Joel T. Boone for the Department of the Interior.

The major consequence of this pattern--the cost externalization, lack of infrastructure development, one-crop economies and undertaxation--takes the form of a tremendous social deficit in the Appalachian coalfields.

Current problems are compounded by the absence of tools to deal with the legacy that carries over from the past. Future occupational and environmental impacts from mining will be added to this existing deficit. In many communities, the margin for additional damage is thin or nonexistent. This implies that even small future increments of environmental degradation will have very large social consequences.

In this context, an overview of coal mining's environmental health implications can be focused in two separate but related areas. First, this discussion will outline some of the direct health impacts of mining on mine workers and environmental health in their communities. Second, a brief discussion is presented of the direct impacts mining has had on the ability of coal communities to cope with current and future occupational and environmental health problems.

DIRECT HEALTH EFFECTS: OCCUPATIONAL AND ENVIRONMENTAL

A. Occupational Safety

Coal mining has never been a safe occupation. Since 1900, mining has killed a recorded 110,000 miners and, since 1930 alone, more than 1½ million disabling injuries have been reported.[1] In 1977, coal operators reported 139 fatalities and 14,933 disabling injuries to MESA.[2] Fatality frequency in 1977 was .36 per million worker hours and disabling injury frequency was 37.77 injuries per million worker-hours.[3] Summary accident data for coal mining in 1977 is presented in Table 1. Trend data for fatalities for 1952-1977 is found in Table 2. Little or no improvement was recorded between 1952 and 1970 in fatality frequency for either underground or surface mining. However, with the implementation of the 1969 Federal

TABLE 1 - Number of injuries, injury-frequency rates per million man-hours, persons working, man-hours, and production, by kind of coal mined and work location, cumulative January-December 1977 1/

Kind of coal and work location	Number of injuries reported to the Mining Enforcement and Safety Administration			Injury-frequency rates 2/ per million man-hours			Average number of persons working 6/	Man-hours reported 3/	Production reported (short tons) 3/
	Fatal	All disabling 4/	All injuries 5/	Fatal	All disabling 4/	All injuries 5/			
Bituminous coal:									
Underground mines:									
Underground------------------	82	10,911	(5,896) 16,807	0.40	52.68	(28.48) 81.16	126,722	203,851,383	254,820,485
Surface----------------------	9	713	(613) 1,326	.38	27.59	(23.76) 51.35	14,541	23,738,735	-
Total, underground mines--	91	11,624	(6,509) 18,133	.40	50.06	(27.99) 78.05	141,263	227,590,118	254,820,485
Strip mines-------------------	26	2,142	(2,296) 4,438	.23	18.12	(19.37) 37.49	61,887	114,104,214	397,722,272
Auger mines-------------------	2	29	(15) 44	1.81	17.17	(8.13) 25.30	1,294	1,106,643	4,256,721
Other surface 7/ -------------	-	2	(2) 4	-	-	(5.04) 5.04	134	198,593	715,580
Total, mining-------------	119	13,797	(8,822) 22,619	.35	39.30	(25.04) 64.35	204,578	342,999,568	657,515,058
Mechanical cleaning plants-----	8	733	(607) 1,340	.27	22.14	(18.89) 41.02	17,670	30,177,926	-
Independent shops and yards----	1	110	(111) 221	.15	16.43	(16.28) 32.71	3,854	6,572,088	-
Total, bituminous coal----	128	14,640	(9,540) 24,180	.34	37.54	(24.40) 61.95	226,102	379,749,582	657,515,058
Pennsylvania anthracite coal:									
Underground mines:									
Underground------------------	9	66	(2) 68	16.28	108.54	(3.62) 112.16	415	552,767	564,787
Surface----------------------	-	1	- 1	-	9.85	- 9.85	74	101,487	-
Total, underground mines--	9	67	(2) 69	13.76	93.24	(3.06) 96.29	489	654,254	564,787
Strip mines-------------------	1	101	(116) 217	.47	43.32	(53.68) 97.00	1,267	2,123,626	2,711,096
Other surface 7/ -------------	-	7	(11) 18	-	30.55	(48.01) 78.57	171	229,106	1,920,484
Total, mining-------------	10	175	(129) 304	3.33	53.21	(42.23) 95.44	1,927	3,006,986	5,196,367
Mechanical cleaning plants-----	1	105	(132) 237	.52	53.95	(68.61) 122.56	1,107	1,909,245	-
Independent shops and yards----	-	13	(29) 42	-	91.48	(204.07) 295.56	76	142,105	-
Total, anthracite coal----	11	293	(290) 583	2.17	54.56	(56.74) 111.30	3,110	5,058,336	5,196,367
All coal:									
Underground mines:									
Underground------------------	91	10,977	(5,898) 16,875	.45	52.83	(28.41) 81.25	127,137	204,404,150	255,385,272
Surface----------------------	9	714	(613) 1,327	.38	27.52	(23.66) 51.17	14,615	23,840,222	-
Total, underground mines--	100	11,691	(6,511) 18,202	.44	50.19	(27.92) 78.10	141,752	228,244,372	255,385,272
All surface mining------------	29	2,281	(2,440) 4,721	.25	18.56	(19.91) 38.48	64,753	117,762,182	407,326,153
Total, mining-------------	129	13,972	(8,951) 22,923	.37	39.42	(25.19) 64.62	206,505	346,006,554	662,711,425
Mechanical cleaning plants-----	9	838	(739) 1,577	.28	24.03	(21.85) 45.88	18,777	32,087,171	-
Independent shops and yards----	1	123	(140) 263	.15	18.02	(20.26) 38.28	3,930	6,714,193	-
Total, all coal-----------	139	14,933	(9,830) 24,763	.36	37.77	(24.83) 62.59	229,212	384,807,918	662,711,425

1/ Information presented in this table is based on data on file February 11, 1978, and is preliminary.
2/ Any injuries from mines that have not reported man-hour data have not been included in the computation of injury-frequency rates.
3/ Based on recorded data from reporting mines; should be used only for the purpose of computing rates; does not constitute industry total.
4/ Includes fatal injuries.
5/ Figures in parentheses are for nondisabling injuries and are included in the accompanying figure.
6/ Summary of average number of persons working for only those months during which each establishment was active (not arithmetic average for all months).
7/ Culm banks and dredges.

SOURCE: MSHA, 1978.

**Table 2—Fatalities in U.S. Coal Mines, 1952-77
(hours of exposure)**

Year	Underground		Surface		Surface fatalities as a percentage of underground
	Fatalities	Rate[a]	Fatalities	Rate[a]	
1952	514	.97	33	.55	6%
1953	440	.96	21	.39	5
1954	374	1.10	22	.48	6
1955	391	1.06	25	.51	6
1956	417	1.11	28	.52	7
1957	451	1.30	22	.42	5
1958	334	1.26	19	.39	6
1959	268	1.11	20	.43	7
1960	295	1.29	24	.52	8
1961	275	1.34	17	.39	6
1962	265	1.34	20	.46	8
1963	257	1.28	22	.49	9
1964	224	1.12	15	.33	7
1965	240	1.21	18	.40	8
1966	202	1.12	23	.55	11
1967	186	1.05	22	.52	12
1968	276	1.63	24	.57	9
1969	163	.95	28	.63	17
1970	220	1.20	29	.55	13
1971	149	.86	23	.39	15
1972	128	.68	20	.38	16
1973	105	.56	16	.28	15
1974	97	.51	24	.33	25
1975	111	.47	32	.33	29
1976	109	.45	23	.22	21
1977	100	.43	27	.23	27

[a] Expressed in million hours of exposure. Data do not include auger mines, culm banks, dredges, preparation plants, shops, and contractors.
SOURCE: Mine Safety and Health Administration, 1978.

Coal Mine Health and Safety Act, the frequency of underground fatalities has been cut by more than half. Fatal frequency in surface mining has also been more than halved. The number of underground fatalities has fallen with the reduction in frequency. However, the number of surface fatalities has increased despite lower frequency as more surface miners were hired in the 1970s. When the data are examined closely, it is apparent that the act has almost eliminated multi-victim underground disasters as a source of fatalities by setting and enforcing standards for methane, combustible dust and electrical equipment. Some improvement has also been recorded in reducing fatalities from roof falls and haulage accidents.

On the other hand, little improvement has been recorded in reducing disabling injuries since 1969. Underground disabling injury frequency was 53.26 per million hours worked in 1952 and 50.86 in 1977, according to data presented in table 3. Surface disabling injury frequency was 28.11 in 1952 and 18.91 in 1977. Overall, disabling frequency for all coal mining has improved from 1952's 50.66 injuries per million worker hours[4] to 37.77 injuries in 1977 (table 1). However, since 1969 little consistent improvement has been recorded.[5] The Act did not address injury prevention specifically.

Table 3—Nonfatal Disabling Injuries in U.S. Coal Mines, 1952-77 (hours of exposure)

	Underground		Surface	
	Injuries	Rate[a]	Injuries	Rate[a]
1952	28,353	53.26	1,698	28.11
1953	22,622	49.38	1,604	29.48
1954	16,360	47.98	1,342	29.01
1955	17,699	48.10	1,140	23.14
1956	18,342	48.88	1,318	24.31
1957	17,076	49.23	1,339	25.72
1958	12,743	47.94	1,150	23.86
1959	10,868	44.90	1,054	22.64
1960	10,520	46.09	1,125	24.45
1961	9,909	48.19	1,052	24.44
1962	9,700	48.88	1,027	23.50
1963	9,744	48.62	1,099	24.25
1964	9,692	48.35	1,116	24.75
1965	9,705	49.09	1,178	26.12
1966	8,766	48.78	1,043	25.00
1967	8,417	47.57	949	22.43
1968	7,972	46.99	1,039	24.59
1969	8,358	48.77	967	21.84
1970	9,531	51.79	1,346	25.67
1971	9,756	56.40	1,564	26.54
1972	10,375	55.32	1,305	24.84
1973	9,206	48.80	1,208	20.94
1974	6,689	34.95	1,229	16.83
1975	8,687	37.13	1,714	17.74
1976	11,390	47.09	2,071	20.04
1977	11,724	50.86	2,246	18.91

[a] Expressed in million hours of exposure. Data does not include auger mines, culm banks, dredges, preparation plants, shops, and contractors.

SOURCE: Mine Safety and Health Administration, 1978.

One measure of injury is "severity," expressed as the number of calendar days not worked by the injured worker because of the injury. On the average each underground temporary total disabling injury resulted in an average of 73 lost calendar days in 1977. The average servity for surface miners was 58 days in 1977.[6] Even more disturbing is the fact that the average severity for temporary total disabling injuries increased dramatically in 1977 over the 1950-1976 experience. In that 27-year period, average underground severity for temporary total injuries was 35 days for underground miners and 26 for surface miners. These averages have been exceeded every year since 1974, suggesting that reported accidents may be of an increasingly serious nature and/or more time is spent recuperating from accidents than in the past--particularly in underground mining. Efforts by coal mine operators, labor and federal enforcement agencies have not succeeded in substantially lowering injury risk.[7]

Estimates of future mine safety fatalities and injuries depend on the level of coal production, the mix of mining methods (surface mining is safer than underground), the number of miners employed, productivity rates and accident frequency.

Most coal supply and demand forecasts predict greatly increased levels of production over the next 20 to 30 years. Considerable disagreement exists over how fast, how much, where and by what methods it will be mined. Most observers say production will double and perhaps triple mid-1970's levels (650 million tons) by 2000; the ratio of surface - to deep-mined production will not be any less than today's 60/40 division; productivity (tons mined per worker per shift) will increase very slowly; and most coal miners will continue to work in underground mines in the East even as the locus of production shifts West. All of these factors will affect coal mine injury and fatality experience. The more workers employed in underground mines, the higher will be industrywide fatalities and injuries, for example.

The Congressional Office of Technology Assessment recently prepared production, employment and injury estimates through 2000. These estimates suggest that coal production is likely to more than double and could possibly triple 1977 tonnage (688 million tons) by 2000. Most production growth will occur after 1985 when production is estimated to better 1977 by between 39% to 66%. No net increase is predicted in production by 1985 for mines east of the Mississippi River, and no increase is predicted for eastern surface mines even by 2000. Almost all of the East's additional production will come from underground operations.

The ratio of eastern to western production is shifting dramatically. In 1977, western production of 166 million tons accounted for about 24% of the nation's total. By 1985 this share is likely to rise to between 46% to 48% of the total, and by 2000, between 52% and 53%. In tonnage terms, western production will increase over 1977 between 299% and 331% by 1985, and by between 506% and 724% by 2000. However, most coal miners will continue to be employed in eastern underground mines despite the massive redistribution to western surface mined production. At both low and high estimates of coal production, underground miners will represent 74% of the total workforce in 1985 and 79% by 2000. Employment is not expected to increase significantly 1985 over 1977's 229,000 workers. By 2000, however, employment estimates range between 337,000 to 487,000 nationwide, depending on the level of coal production.

Probable health and safety costs of increased coal production can be calculated using these estimates and current accident frequency rates. Annual fatality and disabling injury forecasts are presented in table 4. It may be argued that current accident rates overstate future costs since they are likely to improve in the future. Perhaps. But trend data for the last few years suggest that the rate of improvement in fatality frequency has slowed almost to the point of stopping. Disabling injury frequency has not changed much at all. Even if these frequencies are lowered, the number of actual fatalities and injuries may still rise due to higher levels of employment.

Table 4 suggests the probability of a substantially higher number of mine fatalities and disabling injuries in the future as production increases. Fatalities are likely to increase to between 157 and 187 by 1985 (over 1977's 139) and to between 259 to 371 by 2000. Disabling injuries are likely to rise from 1977's 14,933 to between 17,000 and 21,000 in 1985 and between 29,000 and 42,000 in 2000. Barring some unforeseen--and unlikely--breakthrough in mining technology or safety practices, these estimates can be taken as representing a likely range of human costs associated with increasing coal production.

Table 4—Mine Worker Fatality and Injury Estimates

		1985		2000	
	1977	Low	High	Low	High
Surface					
Fatalities	29	25	30	32	46
Injuries	2,281	1,954	2,401	2,515	3,634
Underground					
Fatalities	100	118	140	203	291
Injuries	11,724	13,907	16,513	24,012	34,405
Other Coal workers[a]					
Fatalities	10	14	17	24	34
Injuries	984	1,586	1,891	2,653	3,804
Total					
Fatalities	139	157	187	259	371
Injuries	14,989	17,447	20,805	29,180	41,843

[a] 1977 data include workers in shops and cleaning plants, but not construction workers. Estimated data for 1985 and 2000 include all other coal workers and uses a 10 percent add-on to the total of underground and surface accidents.

SOURCE: Office of Technology Assessment, The Direct Use of Coal: Problems and Prospects of Production and Combustion (Washington, D.C.: U.S. Caongress, 1979), p. 289.

Occupational Health

The most serious occupational health hazard miners face is coal mine dust. Respirable coal mine dust is retained in the gas-exchanging portion of the lungs. Exposure to sufficient quantities of respirable coal mine dust over a period of years can lead to coal workers' penumoconiosis (CWP), which, in its most serious form, is fatal. Additional respiratory impairment may result from one or more of the following: job exposure to "nonrespirable" dust (which affects the upper respiratory tract); toxic fumes and gases (produced as combustion products from fires in mine machinery, conveyor belts, lubricating oils and the like); trace elements (as both respirable and nonrespirable dust particles); and mines gases. Other health hazards include noise, hot and cold environments, stress and whole-body vibration. The most recent mortality study[8] found that coal miners died more often than expected from a variety of respiratory diseases such as influenza, emphysema and asthma. Penumoconiosis and bronchitis were underlying causes of death in a significant number of deaths attributed to nonmalignant respiratory diseases. Cancer of the lung and stomach were also higher, which may or may not be related to work factors.

The 1969 Federal Coal Mine Health and Safety Act phased in a 2 mg./m^3 standard for respirable coal mine dust and established a "black lung" compensation program. This standard is the strictest in the Western world. It is based on British research done in the 1960s. Probability curves developed from the British data suggest that at 2 mg., one to two percent of those exposed to no more than 2 mg. of dust over 35 years would develop simple CWP.[9] Recently reported followup data suggests that the "risks of developing category 2 or more coalworkers' simple penumoconiosis over a working life are one or two percent {probability units} higher than earlier predictions."[10]

The 2 mg. standard may not prove to be as safe over time as the early British research promised. A number of methodological questions can be raised about the original research design and subsequent data manipulations. Methodological criticism may be warranted on the data manipulation of both sides of the dose-reponse relationship."[11] No epidemiological study has been done--or could be done--of miners actually exposed to 2 mg. over a working life due to the newness of the standard. The second round of NIOSH's National Study of Coal Workers' Pneumoconiosis done in 1973-1975 by the Appalachian Laboratory for Occupational Safety and Health (ALOSH a facility of NIOSH) found a 6% rate of disease progression among working miners x-rayed in both rounds.[12]

It is possible--and altogether likely--that future research will prove that the 2 mg. standard is not as safe as originally believed. This implies that CWP will continue in the future even if every mine complies with the 2 mg. standard every working day from now on.

Estimates of future prevalence of CWP can be made using available data. Current NIOSH/ALOSH research suggests that a 10% to 15% prevalence rate among working miners in the early 1970s is justified. Prevalence among working miners in 1979 is probably lower inasmuch as about 40% of miners working in 1969 have retired and almost 150,000 new workers have entered the mines in the 1970s. Table 5 presents a set of CWP estimates associated with increased coal production I developed for OTA. Under optimistic assumptions of diligent dust control, as many as 10,000 miners would show x-ray evidence of CWP in 2000. Under less optimistic assumptions, perhaps 18,000 cases would be detected. If the 2 mg. standard is not as safe as as is currently assumed and if dust control efforts are not diligent, these numbers will be higher.

Table 5 —Estimates of CWP Among Working Miners, 1976, 1985, and 2000

	Prevalence Rates			
	Optimistic		Pessimistic	
	Underground	Surface[a]	Underground	Surface[a]
Working miners	*10%*	*4%*	*15%*	*4%*
1976 (208,000)				
Underground (70%)				
145,600	14,560		21,840	
Surface (30%)				
62,400		2,496		2,496
Total	17,056		24,336	
Working miners	*7%*	*3%*	*10%*	*3%*
1985 (229,829)				
Underground (74%)				
169,922	11,895		16,992	
Surface (26%)				
59,907		1,797		1,797
Total	13,692		18,789	
Working miners	*3%*	*1%*	*5%*	*2%*
2000 (410,893)				
Underground (79%)				
326,305	9,789		16,315	
Surface (21%)				
84,588		846		1,692
Total	10,635		18,007	

[a] Prevalence rates for 1976 are consistent with the range of current research findings. The 4 percent prevalence rate for surface miners comes from R. Paul Fairman, Richard J. O'Brien, Steve Swecker, Harlan Amandus, and Earle P. Shoub, "Respiratory Status of the U.S. Surface Coal Miners," unpublished manuscript done for ALOSH NIOSH, 1976. The ALOSH study used X-ray evidence to determine the prevalence of CWP. Most of the surface miners had extensive experience in underground coal mining which contributed to the prevalence of CWP. Prevalence rates for 1985 and 2000 assume that compliance with the current dust standard will lower prevalence. In addition, increasing numbers of older miners who had years of exposure before 1969 will retire, thereby lowering prevalence among working miners. These prevalence rates have not been derived mathematically, and should be seen more as possibilities than as predictions.

SOURCE: OTA, Direct Use of Coal, p. 265.

CWP is one work-related disease process found in coal miners. "Black lung" is the vernacular name miners use to describe all respiratory disability associated with their trade. Black lung includes, CWP, as well as occupational bronchitis and emphysema, severe dyspnea, airways obstruction and as yet an unnamed disease process affecting the gas-exchaning portion of the lungs.[13] These various diseases may occur simultaneously in a single miner and in different combinations. Cigarette smoking increases the risk of lung disease among coal miners as in the population generally. Some of these disease processes are probably related to nonrespirable coal-mine dust and to the non-coal constituents of that dust (trace elements and quartz). No federal standards exist for nonrespirable dust or for toxic respirable substances encountered in a mine environment. It is reasonable to assume that the prevalence of black lung disability will continue to be significant among coal miners in the absence of controls on these hazards. Emphasis on CWP and respirable dust may have concealed meaningful dose-response relationships between black lung and nonrespirable dust as well as other harmful air-borne substances. Since many more cases of black lung appear today than of x-ray diagnosed CWP,[14] it is prudent ot suppose that a similar ratio will continue in the future. In a rough way it can be said that for every case of CWP, at least two cases of bronchitis, one case of severe dyspnea and one case of airways obstruction will be found, although not necessarily exclusively.

NIOSH calculated future occupational health costs of increased coal production in 1977. These estimates are presented in table 6. NIOSH's prediction of CWP cases are considerable higher than those found in table 5.

Table 6 —Projected Health Effects of Increased Coal Production

Year	Millions of tons produced (quads)	Number of employees	Cases[a] of CWP	Cases of[ab] chronic bronchitis	Cases of[ab] severe dyspnea	Cases of[ab] airways obstruction
1975 estimates						
Underground	279 (7.3)	139,500	18,100	41,800	11,200	41,800
Strip auger	332 (7.9)	52,500	1,300	15,700	4,200	10,000
Total	611 (15.2)	192,000	19,400	57,500	15,400	51,800
1985 estimates						
Underground	395 (9.8)	197,500	25,600	59,200	15,900	59,200
Strip auger	735 (18.3)	116,000	2,900	34,800	9,300	22,100
Total	1,130 (28.1)	313,500	28,500	94,000	25,200	81,300
2000 estimates						
Underground	630 (15.7)	315,000	40,900	94,400	25,300	94,400
Strip auger	1,170 (29.2)	185,000	4,600	66,300	14,800	35,200
Total	1,800 (44.9)	500,000	45,500	160,700	40,100	129.600

[a] Not mutually exclusive.

[b] Not necessarily due solely to coal dust inhalation.

SOURCE: National Institute for Occupational Safety and Health, Occupational Safety and Health Implications of Increased Coal Utilization," computer printout and attachments (rev : Nov. 4, 1977. Distributed at a NIOSH conference of the committee on health and ecological effects of increased coal utilization, Nov. 21, 1977.

NIOSH used a 10% CWP prevalence rate for working miners for 1975, 9% in 1985, and 9% in 2000. These prevalence rates assume the current 2 mg. dust standard will have little beneficial effect on CWP prevalence. NIOSH's assumption may cast doubt either about the standard's inherent safeness or about the extent of compliance. NIOSH also used somewhat higher estimates of the future mine-worker labor force than OTA did (table 5). The OTA and NIOSH estimates represent a band of possible occupational health implications of increased coal production.[15] As a matter of personal opinion, I believe the OTA estimates will prove to be too low and the NIOSH ones too high. The range of likely adverse health effects these two efforts present are: 1985, 13,700 to 28,500 cases of CWP; 2000, 10,600 to 45,500 cases of CWP. Bronchitis, severe dyspnea and airways obstruction should each occur in at least a 1:1 ratio to CWP.

Future prevalence of work-related lung disease among miners will be determined in large part by the effectiveness of dust control measures implemented by mine operators and coal miners. As there was no systematic dust sampling and no standard before 1970, dose-response comparisons between that era and now are difficult, if not impossible. The few dust surveys done before 1970 found dust concentrations far in excess of the current standard. For example Schlick and Fannick reported the results of 1968 Bureau took nearly 2,000 samples and found the mean dust concentrations of the 16 occupation categories sampled exceeded 2 $mg./m^3$.[16]

Miners who have worked in both periods say dust conditions are better now than before. Clearly, the federal standard has forced mine operators to adopt deliberate programs ot control respirable dust. It's fair to say that many operators are making good-faith efforts to keep their working sections in compliance. Others, on the other hand, arent't.

The quantitative measure of this effort lies in the data accumulated from the operator-submitted respirable dust samples submitted to MSHA several (on the average) times a year. Miners performing certain jobs are required to wear a personal sampler for a full shift at specified intervals throughout the year. The samplers are turned into the company at the end of the shift, and the company sends the sample to MSHA for weighing. Several weeks later the results are sent back to the operator. If the sample exceeds the standard, the miner is required to take 10 additional samples until the results indicate compliance.

A number of flaws in this system have been pointed out.

First, the lag-time between when the sample is taken and when the results are received is of great that the sampling has little relevance to daily dust control efforts. Mining conditions change frequently. Without immediate feedback from the monitoring, sampling results are useful mainly as a basis for civil penalties and not for dust control. Second, incentives exist for both miners and operators to falsify sample results. Samplers are noisy and a nuisance to wear, especially in cramped conditions. Many miners believe that operators only submit "good samples" and order retakes of the "bad" ones (that is, those showing excessive dust). Some evidence is available to suggest that this opinion is well-founded. Some miners report that their countries weigh the entire sampler after each shift, rejecting those that are "too heavy." Why, then they ask, should we bother taking samples when the company simply redoes or falsifies "bad" results?" A "bad" sample means the miner is required to take 10 more samples, a prospect that encourages him to go along with falsification. Some miners believe that compliance samples will be used against them in future black lung eligibility determinations, and apparently try to "stuff" the sample to record high dust concentration. MSHA has devised methods to void samples showing extremely high concentrations of nonrespirable dust, which, the agency believes, indicates "stuffing." Coal mine operators, of course, have an interest in submitting compliance samples to MSHA. Such samples minimize operator inconvenience and avoid the possibility of civil fines, which, for respirable dust violations, averaged about $150 in 1978.[17] In one case, two management technicians with dust sampling responsibility at a Consoldiation Coal Co. mine in Ohio were found guilty--and then innocent--of deliberate fasification of sample results. Finally, MSHA has not been able to establish adequate safeguards against sample falsification, which vary from leaving the sampler in the dinner hole and sample stuffing to operator voiding of samples. Samples taken by MSHA's own inspectors are even more infrequent and often subject to the same kinds of distortions.[18] While sample data from operator submissions suggest a 90%+ compliance rate on the days the samples are taken, MSHA inspectors cited 36% of the almost 5,000 underground sections they sampled in 1976 for noncompliance. Further, MSHA issues an average of about 2,500 notices of noncompliance and 50 withdrawal orders annually for respirable dust violations, casting further doubt on 90%+ compliance. As in any other representative survey, dust sampling may or may not be good reflection of dust exposure on the 200 or so days each year when sampling is not done.

Several studies have found such weaknesses in the federal dust sampling program. The General Accounting Office reported:

> "...many weaknesses in the dust-sampling program which affected the accuracy and validity of the results and made it virtually impossible to determine how many mine sections were in compliance with statutorily established dust standards."[19]

GAO said factors that affected sample accuracy were: sampling practices used by operators and miners, dust sampling equipment, weight loss of sampling cassettes, and weighting of cassettes by MESA and cassette manufacture's.[20]

A National Bureau of Standards (NBS) study found:

> ...when the miners and mine operators perform and supervise the sampling and when the weightings are made in the normal manner,... the uncertainty (of accurate, actual measurements of dust concentrations) is estimated to be as large as 31 percent..(Emphasis in the original.)[21]

No follow-up study on the accuracy of the sampling program has been done since the GAO and NBS reports were released in December 1975. Both investigations noted that MSHA and NIOSH officials have made efforts to improve.

A recent study of underground mines in East Kentucky found:

> Both the interview and the federal dust records suggest that many dust samples are being collected incorrectly. In some instances, extra dust has been added; but in substantially more cases, the samples are too low. One explanation for inaccurrate sampling, of course, lies in the fact that the mine owners control sampling in their own mines. Since the penalty for exceeding federal standards may be close to mine, coal operators have a strong incentive to err in the direction of samples showing less than the actual dust levels. The 1969 law attempts to circumvent this possibility by requiring that all samples be collected by actual miners on the assumption that, since their own health is at stake, coal miners will have a vested interest in insuring the accuracy of samples taken at their work place. For various reasons, however, that assumption has proven wrong."[22]

Sharp found 27 percent of the 680 face and high-risk samples he examined were 0.1 or 0.2 mg., which are generally considered to be impossibly low. (Those readings represent dust levels in fresh air.) About 23 percent of equipment operators' samples were also found to be too low to be realistic. Operator attitude toward the sampling program has an important effect on sampling accuracy, Sharp concluded.

These studies as well as anecdotal evidence suggests that the federal dust sampling program is probably not a reliable indicator of daily respirable dust exposures. The implication is that it is possible and probably likely--that many miners are regularly exposed to illegal and unhealthy dust concentrations. It is impossible to say how high daily exposure is in every mine or how often compliance is achieved. From the perspective of occupational health policy, it is unjustified to believe that future prevalence of respiratory disease in coal miners can be based on the assumption that most mines are in daily compliance with the federal standard.

For these reasons and others, MSHA has proposed shifting from personal to area dust sampling. Some of the unreliability in dust data may be eliminated through this change, MSHA believes. MSHA has not proposed that control of the sampling program be altered or that the lag-time problem be addressed. In response, the United Mine Workers (UMWA), has instead proposed to MSHA that a miner-elected, MSHA-trained miner be in charge of sampling at each mine. The union has suggested that area samples be taken by recording dust monitors capable of providing instantaneous printed readouts. These monitors would enable miners and foremen to immediately correct dangerously high exposures. Further, the union has suggested that personal sampling be continued to determine compliance with federal law. Miner control and immediate feedback would, the UMWA says, make sampling more effective in protecting miners' health. The coal industry has opposed area sampling and miner participation. MSHA has not yet reached a final decision on the matter.

While respiratory disease is the most serious job-related health hazard miners face, other exposures--to noise, fumes, gases, stress, whole-body vibration, cramped conditions, hot and cold environments and wet working conditions--also exist. Hearing surveys indicate that miners experience more hearing loss than expected, although the degree to which hearing loss is job related is disputed.[23] Noise levels for certain mining jobs have been found to exceed the 90 dba federal limit. Available noise sampling data is not very accurate, MSHA officials admit. Often, many of the hazards noted above occur simultaneously. Occupational health research has not been able to analyze the long-term effects (including synergisms) of multiple hazards. Future investigation may begin to develop tools for multi-variable analysis. The need for this kind of research is clear: it is how a coal miner is affected by his work.

ENVIRONMENTAL HEALTH EFFECTS OF COAL MINING

Coal mining--as distinct from coal combustion--affects the air, land and water where it occurs. In this discussion, a distinction is drawn between mining's adverse effects on the environment and those on human health stemming from environmental degradation. This overview is limited to the latter.

AIR

Fugitive dust is a problem in some surface mines. Recent federal regulations require mine operators to develop fugitive-dust control plans and implement them.[24] Coal-truck haulage also creates fugitive dust, (as well as significant safety hazards). Since much coalfield housing is squeezed into narrow valley floors adjacent to coal-haul roads, fugitive dust is an important health issue to local residents. Often haulage-dust combines blow-off or spillage from the coal with whatever dust (dirt and coal) is disturbed by traffic flow.

Noxious fumes from burning coal-mine refuse piles are probably mining's most substantial air-borne health hazard. A survey done by the U. S. Bureau of Mines in 1968 found 292 fires burning in coal refuse banks, principally in Appalachia.[25] Most of these--132--were found in West Virginia. Smoke, particulates, toxic and noxious gases are released by burning coal-refuse piles. Gaseous emissions from these waste piles were surveyed in 1968. These data are found in table 7. Coal-waste piles alone generated at least 1% of the nation's total of all carbon monoxide, sulfur oxide, nitrogen oxides and particulates. As these piles are concentrated in the Appalachian coalfields--and usually within steep-sided valleys--their potential health impact on local populations is amplified. More than 90,000 persons, for example, lived within three miles of burning gob piles in West Virginia alone.[26] In 1940, several doctors testified that sulfur dioxide emitted from burning piles located 1-1/4 miles from Triadelphia, W VA., was sufficient to cause severe problems to the town's residents.[27] The author is not aware of any current epidemiological study of respiratory effects associated with burning gob piles. New surface mining regulations require that coal-mine waste fires be extinguished by the mine operator in accordance with an approved plan. Orphan piles may continue to be a source of concern.

TABLE 7

GASES EMITTED INTO ATMOSPHERE FROM BURNING COAL REFUSE BANKS IN 1968

Gas	Emissions (Million Tons)	Percent of Total
Carbon Monoxide	1.2	1.2
Sulfur Oxide	.6	1.8
Hydrocarbons	.2	.6
Nitrogen Oxides	.2	1.0
Particulate	.4	1.4

SOURCE: Nationwide Inventory of Air Pollutant Emissions, 1968 National Pollution Control Administration, AP-73, August, 1970.

Blasting from surface mining is a third air-related hazard. Residents living adjacent to strip mines have been injured by rocks and debris striking them or their homes. In one case, a 1000-pound boulder reportedly flew 50 feet and crashed into a Floyd County, Kentucky house injuring a woman's hand. The Janis Grear Mine paid $4,700 in property damage, but nothing for personal injury in this case.[28] Blasting noise is also a nuisance. Mining noise has been addressed in the final surface mining regulations which established noise limits and for mining and set up other blasting restrictions.

LAND

The two most serious health hazards from mining's impact on land are surface subsidence from underground mining and dangers related to the disposal of coal-refuse.

Subsidence is a familiar problem throughout much of coalfield West Virginia, Illinois and Pennsylvania. When underground mining occurs, pillars of coal are left to support the roof.[29] Over time, these pillars erode and weaken. When deterioration is extensive, the strata above the mined-out area will collapse into the void, lowering portions of the surface by the width of the coal seam or less (a few inches to 6 feet or more). Property damage often results and residents may be injured. A recent survey done by the General Accounting Office reported that mine subsidence results in nearly $30 million in property damage annually.[30] GAO estimates that two of the 8 million acres undermined in the U. S. have already subsided to some degree, and another 2 million acres will subside by 2000 when total undermined acreage reaches 10.5 million unless adequate preventive measures are taken. No reporting system exists to tabulate injuries or pyschological stress related to subsidence.[24A] Subsidence-related injuries are not reported to any federal agency. The always present danger of subsidence impedes the development of non-coal industry and community facilities in the coalfields. The opportunity costs of subsidence are significant. Feasible techniques are available to minimize subsidence by backstowing waste material--either flyash from coal combustion or the coal mine waste itself. Coal operators have not backstowed waste material because of the dollar costs involved.[30A]

Dr. Robert Erwin, head of the West Virginia Geological Survey, said recently that thousands of West Virginian homes are being ruined and lives threatened by subsidence from abandoned underground mines.[30B] "'I wouldn't rule out the chance of a youngster falling into a crevice caused by subsidence in some places.'" A 7-year-old boy was reported drowned in Seneca, Illinois, when "...an abandoned mine shaft filled with water collapsed in his back yard."

Mining produces much refuse, which, in the past, has often been disposed of haphazardly and with little attention paid to environmental or health considerations. This refuse consists of materials extracted along with the coal which are separated and discarded on the surface. (Other refuse is produced from coal washing and preparation, together with sludge from treating acid-mine waters and scrubbing coal's flue gasses.) About 107 million tons of mine refuse was produced in 1975, (table 8). The fraction of run-of-the-mine material discarded in preparation is now about 29%. Many coal consumers are demanding even cleaner coal. More than 3 billion tons of mine waste are estimated to have been piled on the surface over the years in some 3,000 to 5,000 refuse banks.

Apart from degrading surface water quality, the main hazard from refuse piles comes from their physical instability. Erosion and landslides can result when refuse is not graded, compacted and reclaimed. Few engineering or reclamation regulations applied to these banks--known as "gob" piles of "slag" heaps--before the 1970s. In some cases, the refuse was heaped into jerry-built dams to create settling ponds used in coal preparation. Following the collapse of a Pittston Corp.-owned gob dam on Buffalo Creek, W. VA in 1973 that killed 125, a survey found at least 30 other dams to be imminent flood hazards and another 176 to be potential hazards.[31] Federal agencies and coal operators have made an effort to safen these dams since the survey. Existing and new gob dams are now covered by recently promulgated OSM regulations. Existing impoundments are likely to present more of a hazard to human life than those built in the future because of the new engineering requirements. The degree of danger these dams present today is difficult to estimate. Fear of dam collapse is often expressed by citizens living downstream from an impoundment. The lack of housing options in the coalfields inhibits ready evacuation of downstream areas.

Table 8 — Mechanically Cleaned Bituminous Coal and Lignite[a]
(thousand short tons)

Year	Total raw coal moved to cleaning plants	Cleaned by wet methods	Cleaned by pneumatic methods	Total cleaned	Total production	Percent of total production mechanically cleaned	Refuse resulting in cleaning process	Percent refuse of raw coal
1940	115.692	87.290	14,980	102.270	460.771	22.2	13,422	11.6
1945	172.899	130.470	17,416	147,886	577.617	25.6	25.013	14.5
1950	238,391	183.170	15,529	198,699	516.311	38.5	39,692	16.6
1955	335.458	252.420	20,295	272,715	464.633	58.7	62,743	18.7
1956	359.378	268.054	24.311	292,365	500.874	58.4	67,013	18.6
1957	376,546	279.259	24.768	304.027	492.704	61.7	72.519	19.2
1958	320.898	240.153	18.882	259.035	410,446	63.1	61,863	19.3
1959	337.138	251.538	18.249	269.787	412.028	65.5	67,351	20.0
1960	337.686	255.030	18.139	273.169	415.512	65.7	65,517	19.4
1961	328.200	247.020	17.691	264,711	402.977	65.7	63.489	19.4
1962	339.408	252.929	18.704	271.633	422.149	64.3	67,775	20.0
1963	362.141	269.527	19,935	289,462	458.928	63.1	72.679	20.0
1964	388.134	288.803	21,400	310.203	486.998	63.7	77,931	20.1
1965	419.046	306.872	25,384	332.256	512.088	64.9	86,790	20.1
1966	435.040	316.421	24,205	340.626	533.881	63.8	94,414	21.7
1967	448.024	328.135	21,268	349,402	552.626	63.2	98,624	22.0
1968	438.030	324.123	16.804	304.923	545,245	62.5	97,107	22.2
1969	435,356	315.596	19,163	334.761	560.505	59.7	100.593	23.1
1970	426.606	305,594	17,855	323.452	602.936	53.6	103.159	24.2
1971	361.168	256.892	14,506	271.401	552,192	49.1	89,766	24.9
1972	398,678	281,119	11,710	292,829	595,387	49.2	105,850	26.5
1973	397,646	278.413	10,505	288.918	591,737	48.8	108,728	27.3
1974	363.334	257,592	7,557	265.150	603.406	43.9	98,184	27.0
1975	374.094	260,289	6,704	266,993	648,438	41.2	107,101	28.6

[a] U.S. Bureau of Mines Yearbook, various years

WATER

Mining can seriously degrade the quality of ground and surface water in mining areas. In addition, some evidence shows that stream siltation in Appalachian watersheds caused by surface mining has increased the threat to human safety and health from flooding.

Ground waters, which are often tapped for residential purposes including drinking, can become contaminated from mining activity. When sulfur-bearing coal is exposed to air and water, a chemical reaction occurs forming sulfuric acid and iron. Heavy metals locked into the coal as trace elements tend to dissolve in the lowered pH, adding metal ions such as aluminum, manganese, zinc and nickel to the drainage. Acid mine drainage can contaminate ground water when it seeps into acquifiers via joints and fractures in the rock or through direct interception of the aquifer.[31A] When a mine is above the water table, acid seepage can occur. When below, water can drain into the mine and lower water tables and dry up wells. Mine-related blasting can dislocate aquifiers. Mining and reclamation can change the permeability of surface soil and rock strata, and alter the rate of groundwater replacement.

Acid mine drainage is a problem more commonly associated with surface waters. The National Strip Mine Study prepared by the U. S. Army Corps of Engineers in 1974 estimated that more than 10,000 miles of Appalachian streams were significantly affected by mine drainage.[22] About 6,300 miles of Appalachian streams were continually polluted by acid drainage, representing about 93 percent of the Nation's total.[33] Northern Appalachia contains many of the polluted waterways. Acid drainage from underground operations--many of them abandoned--accounts for pollution in the Basins of the Monongahela, Allegheny, Susquehanna, and North Branch of the Potomac. The Environmental Protection Agency reported almost 20 percent of the total stream miles in the Monogahela Basin are polluted by acid drainage.

Acid surface water can be unfit for human consumption, expecially when concentrations of heavy metals--some of which are carcinogenic--are high.[34] As water treatment and central water systems are not well-developed in many coalfield areas, the health hazards of contaminated drinking water are significant for many families. Compliance with recent regulations should minimize acid drainage from operating mines, but abandoned mines will continue to create acid drainage.

In mountainous regions, surface mining has frequently created substantial siltation in adjacent watersheds. Before regulations were in effect, operators often pushed unwanted soil, called "overburden" or "spoil," down the mountain side. Rain eroded these unstable slopes causing landslides and stream siltation. Research in Kentucky indicates that sentiment yields from strip-mined lands were as much as 1,000 times that of undisturbed forests.[35] In the Middle Atlantic states, on the order of 30,000 tons per year of suspended solids have been attributed to surface mining and 17,500 tons per year in the midwest. Reclamation of abandoned strip mines and compliance with OSM regulations in active operations should reduce this impact. However, since much future siltation from mining will occur in watersheds already heavily loaded by past mining practices, the margin available for additional siltation may be very small, particularly in many narrow Appalachian valleys.

Flooding, of course, is the principal danger from heavily silted streams in that area. Much controversy exists over whether strip mining increases surface runoff and amount, thereby silting stream channels and increasing flooding potential and danger. Industry argues that surface mining often makes affected land more permeable, reducing runoff and decreasing stream siltation.

Steep terrain, forest fires, heavy rainfall, housing, roads and other construction near natural stream beds are the principal causes of flooding in Appalachian watersheds, industry argues.[36]

Most state or Federally funded studies, however, suggest that surface mining (as practiced before the 1977 law was enacted) contributes to stream siltation and to flooding. The Kentucky Department of Natural Resources, for example, studied the 1977 floods in East Kentucky and found:

> The preponderance of evidence from previous studies indicates that active, unreclaimed or improperly reclaimed surface mined areas increase runoff.[37]

The claim that surface mining decreased flooding by increasing the porosity of the affected land was challenged by Drnevich et. al:

> One of the more startling findings was that the pereability of the mine spoils was about four orders of magnitude lower than what might be predicted based on particle size distribution curves. This low pereability was attributed to the breakdown and weathering of the soil during the compaction and wetting processes.[38]

A recent case study of flooding in Harlan County, KY, acknowledged that road construction, heavy rainfall and other factors contributed to the devastating floods there in 1977, but found that these factors are "...small in comparison to surface mining."[39] Detailed examination of local watersheds, Hardt says, indicates that "...surface mining has had a significant effect on flooding in the county."[40] This study suggests that surface mining increased the April 1977 flood level at Harlan at least three feet and was responsible for at least $3 million of the damage from the April flood.[41] A preliminary estimate of the dollar costs of the 1977 flooding in East Kentucky was $175.1 million.[42]

Apart from the dollar costs incurred by public agencies and private interests,local residents are killed, injured and psychologically affected by flooding. In the 15 East Kentucky counties flooded in 1977, the Red Cross found 10 flood-related fatalities, 2,255 persons injured or made ill, and 132 persons hospitalized. More than 9,700 families suffered property losses and more than 4,500 homes and businesses were completely destroyed or suffered major damage.[43] Several investigators have reported emotional traumas caused by the Buffalo Creek disaster in Logan County, W VA.[44] Children and adults who have experienced flood trauma spend a great deal of energy and time reconstructing their lives and coping with the stress of readjustment in later years. Fears, phobias, sleeplessness, nervousness, irritability, fatigue, physical illnesses (headaches, backaches, ulcers), depression, marital tensions, and alcoholism have been identified as flood-caused legacies among survivors.

II. Mining's Indirect Effects on Health Care Capabilities in the Coalfields

The impact of occupational disease and injury and the impact of environmental health hazards is determined in part by the facilities and services that exist locally which are capable of preventing these problems and treating them when they occur. This capability consists of a network of health-related institutions, personnel, services and perspectives that plays an active role in both prevention and treatment. Often, in coalfield Appalachia, a high level of adverse health impact coexists with an underdeveloped infrastructure for prevention and care. Both sides of the balance sheet are shaped by the general quality of life--living conditions, housing stock, personal income, community development and so on--that is found.

Some of these factors in Appalachia have changed markedly over the last decade, while others remain the same.

Substandard living conditions and inadequate public services have existed in coalfield communities for decades. Journalists, novelists and social scientists have all reported the dimensions and subtleties of the problems there.[45] It is also true, of course, that some conditions have changed for the better within the last decade. Coal miners who work a full year can earn $20,000 or more.[46] The rise in coal prices that began in 1970 and increased spectacularly in 1973-1974 following the OPEC embargo made rich dozens of coal entrepreneurs. Federal disability benefits for black lung victims and higher UMWA pension benefits. Spendable income in the coalfields has risen since 1970 because of higher wages. The depression that beset Appalachia throughout most of the 1950s and 1960s ended for many working miners and their families, although this region continues to have a sizeable population of low- and fixed-income citizens.

Yet, as in many countries of the Third World, indices of economic growth such as personal per capita income do not necessarily reflect proportional gains in public economic development (measure in terms of adequacy of community services and institutions, public health, and long-term economic security). The decoupling of economic growth from economic development in the Appalachian coalfields may have significant consequences in the long-term future as coal resources are extracted and economic diversification does not occur.

The role of coal production is central to the development of under-development in Appalachia. Poor market conditions and cut-throat competition squeezed the coal industry for most of this century leaving as a by-product severe under-capitalization of private and public infrastructures in communities there. Low wages and the company-town system prevented miners from purchasing and building adequate housing. The lack of effective taxation on coal production and undeveloped minerals prevented coal towns and counties from developing good schools, water and sewage services, recreational facilities and many other public services.

Absentee ownership of coal resources and coal companies drew profits away from the communities where they were made, leaving them starved for capital. The most recent study of Appalachian capital needs found that energy development is "...likely to be the source of about one-fourth of Appalachia's public investment shortfalls ...somewhat less than $200 million..." during the 1977-1985 period.[47] Total private investment requirements to support adequately Appalachian energy development will total about $6 billion, of which about 25% will be demanded from local financial institutions.

This report said:

> Serious shortfalls are likely to occur in the availability of private financing for housing... and ...overall capital problems are most severe in the coal mining areas of eastern Kentucky, the Central Appalachian portion of West Virginia, southwestern Virginia and eastern Tennessee where the need is created by nuclear power-plant construction rather than coal development.
>
> ...past deficits in public facility provision and maintenance are not likely to be overcome by future Appalachian energy developments. In some significant cases, new Appalachian energy development will not generate enough revenue or tax base--in the right places--to cope with near-term energy accommodation problems.[48]

For these reasons and others, diversified economies never developed in much of the Appalachian coalfields, thus chaining them to the whimsies of coal's boom-bust cycle and protracted demand stagnation. Today, the consequences of these patterns are apparent. Public services--roads, schools, medical care, governmental capability--are frequently inadequate leaving communities incapable to taking advantage of the benefits of future coal growth. Where coal mining dominated local economies, the social and economic problems seem more pronounced and the capability of responding to growth less evident. Several recent studies have described the connection between concentrated land ownership and inadequate taxation revenues on one hand with extensive community needs on the other.[49]

These larger social and economic patterns determine the kind of public health measures and treatment capability available to coalfield residents. For example, absence or inadequacy of water and sewage treatment facilities is an important factor in preventive health. In Raleigh County, W VA, one public health official told an interviewer from the University of Kentucky that the only thing standing in the way of a typhoid epidemic breaking out in some sewage-befouled hollows was the acid mine drainage that killed the bacteria.[50]

The medical plan negotiated between the United Mine Workers of America (UMWA) and the Bituminous Coal Operators' Association in 1978 instituted co-payment on physician care. This change is likely to reduce doctor visitations and preventive care. The UMWA Funds have also ended negotiated retainer relationships with several dozen miner-oriented clinics and hospitals, forcing them to cut services.[51] Although West Virginia has begun to increase the assessed value of undeveloped coal property, it is doubtful that this will be able to produce enough tax revenue to help this network of health providers. Coal companies are challenging higher tax bills in several counties.

Conclusion

Most observers feel that coal production will increase steadily over the next 20 to 30 years, although demand limitations for certain kinds of coal will continue intermittently. In light of this radical break with coal's past, it seems imperative to mine coal differently than before. Many externalities have been regulated by federal legistlation since 1969. But the adequacy of some current occupational and environmental standards are questioned, and the degree of compliance and enforcement is moot. Industry has not accepted occupational and environmental regulation gracefully. A spirited, well-financed campaign against "excessive" regulation fills the coalfield media. The National Coal Association recently submitted to the White House a 32-page list of federal policies and regulations it wanted changed to unleash coal production and combustion.[52]

The patterns of the past need not--and should not--be repeated as coal demand improves. Five general principles related to occupational and environmental health can be set forth to guide responsible coal development. First, production costs--occupational, environmental and social--should be fully internalized. Second, it follows that coal prices--as well as the price of other fuels--should not be artifically low and should reflect the true costs of the product. Third, a different social calculus for apportioning economic benefits between coal developers and local communities needs to be negotiated. Fourth, momentary disruptions in oil and gas supplies should not be the pretext for turning the coalfields into a national sacrifice area through relaxing necessary occupational and enviornmental regulations on coal. Finally, a wide spectrum of local citizens should participate in planning local coal development--its extent, pace and conduct. In the past, these decisions were made by private interests concerned only with their own ratios of costs to benefits. Since mining scatters many kinds of costs and benefits in local communities, those affected have a right to participate in the economic and political decisions that shape the quality of their lives.

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[2] Mine Safety and Health Administration "Coal-Mine Injuries and Worktime," 1978, p. 4.

[3] When MESA was transferred from the Department of the Interior to the Department of Labor in the spring of 1978, it was renamed the Mine Safety and Health Administration (MSHA). The insertion of "health" into the agency's name may indicate a renewed concern for the occupational health problems in mining.

[4] MESA, Injury Experience in Coal Mining, 1975, p. 125.

[5] Ibid., The 1969 disabling freqency rate for all coal both underground and surface was 41.76.

[6] Data supplied by MSHA.

[7] It should be added, however, that the federal accident reporting requirement was tightened in the 1970s so trend data may conceal real improvement. On the other hand, it is difficult to estimate accurately the extent of underreporting and undercounting of accidents. Tighter reporting requirements may actually stimulate less accurate reporting. Numerous other weaknesses in the federal data harass those who have tried to make sense of it.

[8] Howard Rockette, Mortality Among Coal Miners Covered by the UMWA Health and Retirement Funds, (Morgantown, W.VA: NIOSH/Alosh, 1977).

[9] M. Jacobsen. S. Rae, W.H. Walton, and J.M. Rogan, "New Dust Standards for British Coal Mines," Nature, Vol. 227, August 1, 1970, p. 447.

[10] M. Jacobsen, "Dust Exposure and Penumoconiosois at 10 British Coal Mines," a paper presented at the Vth International Pneumoconiosis Conference, Caracas, Venezuela, October 2 - November 3, 1978.

[11] See OTA, The Direct Use of Coal for a brief discussion of some of these questions. Also James Weeks, "Review of Scientific Data for the U.S. Standard for permissible Exposure to Coal Mine Dust," OTA contractor report, November 1, 1978.

[12] Telephone interview with Harlan Amandus, ALOSH, April, 1979. Fifty to sixty percent of the miners X-rayed in round 1 were not X-rayed in round 2. Progression in those who were not X-rayed in round 2 is likely to be considerably higher given the high rate of mine-worker retirement in these years. ALOSH has not tried to evaluate the reliability of MSHA's dust samples, the majority of which indicate compliance with the 2 mg. standard.

[13] Donald L. Rasmussen, "Breathlessness in Southern Appalachian Coal Miners," Respiratory Care, Vol. 16, No. 2, March-April, 1971.

[14] The 1969 Coal Act--PL 91-173, Title IV, Part A-- provides "black lung benefits" to "coal miners who are totally disabled due to pneumoconiosis and to the surviving dependents of miners whose death was due to such disease." "Black lung benefits" are awarded to victims of penumoconiosis, which is defined as a "chronic dust disease of the lung arising out of employment in an underground coal mine." (Surface miners and othe coal workers are also eligible for benefits.) Between 1970 and December 31, 1977, 421,000 compensation awards were granted by the Social Security Administration and Department of Labor. This represents a number equivalent to the entire coal-miner workforce in 1950. Obviously, the number of awards for exceeds the prevalence rates established for CWP by epidemiological surveys and analysis. This suggests that federal officials have defined compensable CWP to include "black lung disease." It may also imply that the accepted CWP prevalence rates are far too low. And it may imply that the eligibility standards have been stretched to include a very wide range of lung impairments some of which undoubtedly, are made worse by cigarette smoking.

[15] Both NIOSH and OTA excluded retired miners from their calculations. If retired miners were included, the number of CWP cases would increase greatly.

[16] Donal P. Schlick and Nicholas L. Fannick, "Coal in the United States," in Marcus M. Key, Lorin E. Kerr and Merle Bundy, Pulmonary Reactions to Coal Dust (New York: Academic Press, 1971), p. 21.

[17] Telephone interview with Larry Beaman, Office of Assessments, MSHA, February, 1979.

[18] For example, MSHA inspectors are often made victims of what is known as "the penetration game." This occurs when a sample is being taken--either by the worker or by the MSHA inspector--on a continuous miner machine. When a foreman orders the operator to take abnormally shallow cuts into the coal face, exposure to respirable dust will be lessened considerably. However, since this technique--the penetration game--reduces production, it is only used when samples are taken, miners and MSHA inspectors say. MSHA inspectors do not have authority to order deeper--that is, normal cuts.

[19] General Accounting Office, Improvements Still Needed in Coal Dusting-Sampling Program and Penalty Assessments and Collections, December 31, 1975, p. 1.

[20] Ibid., p. 15.

[21] National Bureau of Standards, An Evaluation of the Accuracy of the Coal Mine Dust Sampling Program Administered by the Department of the Interior, A Final Report to the Senate Committee on Labor and Public Welfare. (Dept. of Commerce, Washington, CD, 1975, p iii.

[22] Gerald Sharp, "Dust Monitoring and Control in the Underground Coal Mines of Eastern Kentucky," Masters Thesis, University of Kentucky, November 1968, p. 4.

[23] NIOSH, Survey of Hearing Loss in the Coal Mining Industry (Washington, D.C.: HEW/NIOSH, 1976); J. A. Lamonica, R.L. Mundell and T. L. Muldoon, Noise in Underground Coal Mines, (Washington, D.C.: Interior/MESA, 1971); and Thomas G. Bobick and Dennis A. Giardino, The Noise Environment of the Underground Coal Mine (Washington, D.C.: Interior/MESA, 1976).

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[25] U. S. Bureau of Mines, Coal Refuse Fires, An Environmental Hazard (Washington, D.C.: Interior/USBM, 1971).

[26] *Ibid.*, p. 21.

[27] From trial records, briefs of counsel, The Board of Commissioners of Ohio County v. Elm Grove Mining Co., 122 W. Va. 422, 9 S.E. 2nd 20, 813 (1940).

[28] "Conference Held on Strip Mine Blasting," Mountain Life and Work, May 1978, p. 27. A report of the proceedings will be available in June, 1979 from Appalachian Science in the Public Interest, Corbin, Ky.

[29] The exception to this pattern occurs when longwall mining systems--which employ controlled subsidence as part of the extraction process--are used. Longwall mining accounts for only 4% of total underground production, however.

[30] General Accounting Office, Alternatives to Protect Property Owners from Damages Caused by Mine Subsidence, February 14, 1979. Much of the GAO's data was derived from a 1977 contractor's report prepared for HUD.

[30A] Appalachian Research and Defense Fund, Disposing of the Coal Waste Disposal Problem (Charleston, W.VA: Appalachian Research and Defense Fund, 1973).

[30B] Erwin quoted in Steve Mullins, "Sinking Mine Tunnels Plague Homes in W.VA," Charleston Daily Mail, January 15, 1979.

[31] U. S. Corps of Engineers, Department of the Army, The National Strip Mine Study, Vol. 1, Summary Report, 1974.

[31A] OTA, Direct Use of Coal, p. 237.

[32] U. S. Army Corps of Engineers, The National Strip Mine Study (Washington, D.C.: Department of the Army, 1974).

[33] Appalachian Regional Commission, "Challenges for Appalachia: Energy, Environment and National Resources" (Washington, D.C.: Appalachian Regional Commission, 1976), p. 497.

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[38] Vincent P. Drnevich, G. Perry Williams and Ronald J. Ebelhar, "Geotechnical Properties of Some Eastern Kentucky Surface Mine Spoils," Proceedings of the Ohio River Valley Soils Seminars,Lexington, Kentucky, 1976.

[39] Jerry Hardt, "Harlan County Flood Report," (Corbin, Kentucky: Appalachia--Science in the Public Interest, 1978), p. 42.

[40] Ibid., p. 4.

[41] Ibid., p. 47.

[42] Al Riutort, Randall Lawrence, and Janet C. McCarty, "Southeast Kentucky's April 1977 Floods: The Cost of Recovery" (Frankford, Kentucky: Kentucky Development Cabinet, Governor's Economic Development Commission and the Appalachian Regional Commission, 1977), p. 2.

[43] Ibid., p. 47.

[44] See, for example, Kai Erikson, Everything in Its Path (New York: Simon and Schuster, 1976).

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[51] One recent study of five clinics by the West Virginia University Department of Community Medicine found that the financial conditions of the clinics worsened measurably with the imposition of co-payments. As utilization fell, receivables rose. Operating deficits were recorded in each quarter studied. Physican staffing fell by 42% from July, 1977 through September, 1978. Non-physician staff declined by 25%. Special preventive health programs were eliminated. See Department of Community Medicine, (West Virginia University), William Kissick, and American Health Management and Consulting Corp., West Virginia Primary Care Study Group: Problems of Reimbursement, November, 1978.

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RESPIRATORY PROBLEMS IN COAL MINERS

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RESPIRATORY PROBLEMS IN COAL MINERS

An increased prevalence and higher mortality from bronchitis and other chest diseases were noted in certain occupations including coal mining by Thackrah and Greenhow in the nineteenth century.[1 & 2] The term bronchitis as used at that time was nonspecific, and in the case of coal miners undoubtedly covered a variety of diseases including coal workers' pneumoconiosis (CWP), silicosis, emphysema, tuberculosis, and bronchitis. It was the advent of the chest X-ray and clinical pulmonary physiology, in conjunction with better pathological and bacteriological techniques, which made it possible to define and sort out the relative contributions of the various diseases to the morbidity and mortality experienced by coal miners.

As a result of numerous epidemiological studies which have been carried out in almost every coal mining country, a clearer definition of the respiratory problems seen in coal miners has emerged. It is apparent that the long continued inhalation of coal dust in high concentrations may lead to two conditions, namely coal workers' pneumoconiosis (CWP) and industrial bronchitis.[3] CWP is best defined as the inhalation of coal dust in the lungs and the tissue's reaction to its presence. It can be divided into two forms - simple and complicated, which is often known as progressive massive fibrosis (PMF).

COAL WORKERS' PNEUMOCONIOSIS

a) Simple

Simple CWP is response to the deposition of coal dust in the lungs.[4] It is recognized by a suitable history of exposure, usually at least 10 years underground, and by the presence of fairly distinctive radiographic appearances. Pathologically it is characterized by the coal macule. This is a stellate aggregate of dust situated round the respiratory bronchiole. With prolonged and severe coal dust exposure, the body's defences are overwhelmed and dust starts to accumulate around the second division of the respiratory bronchiole. This is accompanied by a little reticulin and occasionally minimal collagenous fibrosis. Later the smooth muscle in the bronchiolar wall atrophies and the bronchiole dilates. The latter is often referred to as focal emphysema and does not extend to the alveoli or major gas exchanging surface. Simple CWP is divided into categories 1, 2 and 3 according to the profusion of small opacities in the chest film. Postmortem studies have shown an excellent relationship between the coal contents of the lung and radiographic category. Physiologically the high grades of simple CWP may lead to certain very minor pulmonary function impairments. Thus the distribution of inspired gas may be slightly disturbed, and the residual volume and alveolo-arterial gradient minimally increased. Static lung compliance sometimes shows an increase and in a few instances the diffusing capacity and the pulmonary arterial tension for oxygen may be slightly reduced. These impairments are usually only seen in categories 2 and 3 simple CWP, are unassociated with symptoms and cannot be regarded as disabling.

The importance of simple CWP lies in the fact that it is a precursor of the development of complicated CWP or progressive massive fibrosis (PMF). The latter occurs on a background of category 2 or 3 simple CWP and if simple CWP can be prevented, it follows there will be a decline in the incidence of PMF. Simple CWP is unassociated with an increased death rate or disability and is innocuous in itself.

b) Complicated CWP or Progressive Massive Fibrosis (PMF)

This is a different and more serious form of CWP.[4] Unlike simple CWP, PMF is a response to coal dust plus some other factor or factors as yet unknown, but possibly immunological. Necessary for its development is a suitably heavy coal dust burden in the lungs. It will, however, only develop when the lung has been suitably primed, that is to say when category 2 or 3 is present. Unlike simple CWP, PMF may develop and progress after dust exposure has ceased. PMF is characterized by the development of a large opacity greater than 1 cm in diameter, plus the other changes of simple pneumoconiosis already alluded to. The large opacity may slowly enlarge and in some instances other large opacities develop elsewhere in the lungs. The earlier a large opacity appears the greater the likelihood that it will progress and cause disability and premature death.

The masses seen in PMF are partly collagenous but the centre is often composed of calcium phosphates and glycosoaminoglycans, various proteins including hydroxyproline, with only about 20 to 30 percent of collagen.[4] As the masses increased in size they obliterate the vascular bed and airways. PMF is subdivided into stages A, B and C according to the size of the large opacities. A is between 1 and 5 cm in size, B between 5 cm and a third of a lung field, and C greater than a third of a lung field. Stages B and C are associated with respiratory impairment and the latter leads to decreased longevity. Physiologically PMF is characterized by a reduction in lung volumes and in the diffusing capacity. The conglomerate masses also lead to ventilation perfusion mismatching and a concomitant increase in the alveolo-arterial gradient for oxygen which may be associated with arterial desaturation. Pulmonary hypertension develops in stages B and C. Airways obstruction is frequent and occurs in the absence of cigarette smoking or concomitant emphysema.

The prevalence and incidence of simple CWP are related to a number of factors of which the most important is exposure to high concentrations of respirable coal mine dust.[5] Long term studies by the National Coal Board of Great Britain and by Reisner in Germany have done much to delineate the risk for various levels of exposure.[6 & 7] Jacobsen has looked into the pneumoconiosis attack rate and likelihood of progression for various dust levels and has been able to show that provided the dust levels are kept below 4.5 mg/M^3, less than 4 out of 100 miners starting off as category 0 will develop category 2 or above over a period of 35 years.[6] Similar findings have been published by Reisner.[7]

Aside from dust, individual susceptibility is important in the development of CWP. Not all miners who are exposed to the same dust levels develop CWP. In addition there are marked regional differences in the prevalence of CWP which cannot be accounted for by the differences in dust exposure when the latter is measured gravimetrically. Whether the difference in prevalence rate is related to the chemical composition or rank of the coal mine remains unknown but there is some evidence in favor of this hypothesis. Probably more important is the propensity for certain coals to fragment into many smaller particles of around 1 micron in size. Thus the mining of certain coals may lead to a higher proportion of larger particles between 4 to 6 microns, while other coals may fragment more easily and lead to a greater proportion of smaller particles of between 0.5 and 2 microns. The latter are recognized to be more dangerous as far as the development of CWP is concerned. Moreover, when considering an eight hour dust measurement, it is important to bear in mind that an almost infinitely greater number of small particles, e.g. around

1 micron in size as compared to particles of 5 to 5 microns need to be present to constitute the same gravimetric measurement.

Dust control is important in that it prevents the development of CWP. Thus were it possible to prevent categories 2 and 3 simple CWP, then PMF would be virtually eliminated, and it is for this reason that dust control is essential. Nonetheless, no amount of dust control will lessen the effects of cigarette smoke-induced disease, and it is pertinent to bear in mind that the latter is still the main cause of respiratory disability in coal miners.

INDUSTRIAL BRONCHITIS

For many years it has been realized that coal miners have a greater prevalence of cough and sputum than does a comparable population of non-miners.[8] Furthermore, most studies have shown that miners tend to have a slightly lower ventilatory capacity than do non-miners. This reduction in ventilatory capacity cannot be accounted for by the presence of pneumoconiosis and until the last few years the explanation remained obscure. Thus, epidemiological studies have failed to show a relationship between increasing category of simple CWP and a decline in ventilatory capacity. Moreover, since there is a relationship between X-ray category and the dust content of the lungs, it can be inferred that the decrease in ventilatory capacity that is found in miners cannot be explained by dust deposition, at least until PMF develops. These apparent anomalies may be explained by two hypotheses:

a) That coal miners develop a form of airways obstruction which is related to their occupation and dust exposure, but which is distinct from the bronchitis that is induced by cigarette smoking. The cause of this airways obstruction could be either emphysema or bronchitis.

b) That differential migration accounts for the differences. Thus were the more fit to leave coal mining within the first five or so years, those who remain would have decreased pulmonary reserve, and this might account for the lower ventilatory capacity observed.

The second hypothesis is considered first since it has been refuted. Most studies have shown that it is not the fitter entrants who leave coal mining, but the less fit and those with respiratory symptoms. The more fit the man in general, the more likely he is to continue coal mining.

The first hypothesis therefore become more likely, and there is much circumstantial evidence in its favor. There are a number of arguments which have been marshalled against the suggestion that emphysema occurs more commonly in coal miners, and these have been alluded to elsewhere.[8] The hypothesis that coal mining induces a nonspecific form of bronchitis is far more attractive. Such a hypothesis would explain the observation that there is often a poor relationship between the symptoms of bronchitis and CWP. Without dwelling unduly on the methodology of several studies which indicate that industrial bronchitis is the culprit, long-continued dust exposure has been shown to lead to cough and sputum and also to decreased ventilatory capacity.[8] The airways obstruction which occurs in industrial bronchitis predominantly arises from involvement of the larger or central airways. Furthermore, there is further evidence to suggest it is the deposition of larger particles in these airways that leads to the mucous gland hypertrophy and excessive mucous secretion. Radiographic stigmata are not seen since the particles deposited in the dead space are removed by the mucocilliary escaltor. This entity which is a result of long continued deposition of dust in the dead space is best referred to as industrial bronchitis and does not lead to the development of emphysema. Finally it must be borne in mind that lowering of dust levels when such standards are measured in terms of respirable dust will not necessarily affect the prevalence of industrial bronchitis, since most of the evidence suggests that the nonrespirable fraction of dust between 5 and 10 microns is more likely responsible for the induction of industrial bronchitis than is the respirable fraction of between 0.5 and 5 microns.

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URANIUM MINING - OCCUPATIONAL AND OTHER HEALTH PROBLEMS

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URANIUM MINING

OCCUPATIONAL AND OTHER HEALTH PROBLEMS

DR. JOSEPH K. WAGONER

It is appropriate to use as an analogy from the history of the develop- of uranium bearing ores as an energy source, to look at it as a model for how we should proceed in the future with regard to health research in relation to energy technologies. I think it is a fair statement to say that respiratory disease problems of underground miners have been with us since ancient times. The first indication that there might be problems with uranium-bearing ores dates from about 1546 when major respiratory disease problems occurred in miners in the Hartz Mountains of central Europe. The specific nature of the respiratory disorder, however, wasn't suspected until about 1887 when the first clinical evidence emerged that the disorder was a malignancy. In 1913, about 30 years later, Arnstein reported that of 665 miners in the Schneeberg area who died during the period of 1875 through 1912, 276 or 40% of them died of lung cancer. In 1932, the physicians in Joachimsthal reported that of 17 deaths among their miners in a one-year period, 53% were due to lung cancer.

Now these investigators noted three characteristics of the disorder. One, the long latent period between the onset of mining and death due to lung cancer; two, the pathologic absence of silicosis; and three, the preponderance of undifferentiated small-cell carcinoma in the histology.

They concluded that the most probable cause of these tumors was the radon present in the air of the mines. They also made note at that time that the miners themselves had stated that discovery of a rich uranium vein was always followed some years later by a strongly increased mortality among themselves. By the 1940's when large-scale mining and milling of uranium bearing ores started with the United States, for nuclear weapons initially, this lung cancer experience and its probable relationship to radioactivity within the mines was fairly well accepted by the independent scientific community, but little heeded by government or industry.

As a result the Public Health Service in the early 1950's initiated a major epidemiologic program to delineate the magnitude of hazards associated with health problems indigenous to the uranium-producing industry. During the period of the 1950's and 1960's medical examination teams in the Colorado plateau area of the United States examined about 5,370 miners and millers. It was in the population of miners that as early as 1962, Archer, Baldwin, and Cooper showed a statistically significant excess of lung cancer among the subset of those uranium miners who had at least three or four years of underground experience. In reviewing the study of the methods of this study at the NIH in 1962, Dr. Brian MacMahon stated, "If after the European experience there were any doubts as to the implication of radioactivity in the etiology of lung cancer, they surely have been dispelled by the findings to date." Dr. MacMahon further stated that the data therefore gave an indication that the problems of the American uranium miners could become a medical disaster that in relative terms could be as great as the European experience, because of the larger population at risk.

In 1964, however, the Surgeon General's Advisory Committee on Smoking and Health concluded that, "Although the induction of lung cancer by radionuclides is probable in man, the evidence is certainly not as firm as in animals." It was in that same year that I and other members of the Public Health Service reported a tenfold increased risk in lung cancer among long-term underground miners and this excess could not be related to age, smoking, nativity, heredity, urbanization, self-selection, diagnostic accuracy or other variables in the mine. Furthermore, for the first time we were able to show that the mean cumulative radiation dose of the uranium miners with respiratory cancer was significantly greater than a matched control, matched on the basis of age, year of initial examination and race and also other kinds of non-respiratory disease. In 1964, Dr. Saccomanno of Grand Junction reported that the lung cancer among the uranium miners, in contrast with controls matched for age, smoking and residence, showed a peculiar distribution with regard to histologic types. They were predominantly undifferentiated small-cell carcinomas that occurred in the more proximal regions of the lung. In 1965 we reported the demonstration of an exposure-response relationship between air-borne radiation, as measured by the term "cumulative working level months," and we demonstrated that this exposure-response relationship persisted even after adjusting for cigarette smoking.

Now on the basis of these and other data the Department of Labor in 1967 issued an order under the Walsh-Healy Public Contracts Act to limit the maximum permissible exposure to uranium miners to 3.6 working level months during any 12-month period, or exposure limit of 0.3 working level in the mines. This standard was to go into effect sometime in January, 1971. However, as has happened in the past as well as present, this proposed stricter standard led to a controversy, to say the least. The Public Health Service and the Department of Labor were opposed by those groups both within and without the Government interested in the continued, uncontrolled exploitation of uranium ores. This debate and controversy stemmed from two aspects of the data. First was that at that time there were no data to demonstrate conclusively that anyone exposed to a few working levels or less had any problem. Of course, there were as yet no populations studied that experienced that limited exposure rate. The other aspect was that the early studies had not demonstrated an increased risk of lung cancer among the non-white miners, nearly all American Indians, a group who because of their cultural, economic or religious reasons, used very little tobacco. These findings led some to speculate at that time that the induction of bronchogenic cancer by radiation in the absence of cigarette smoking was either very unlikely or nonexistant.

It was speculation on those points that naturally led those with a special interest in the continued uncontrolled exploiting of uranium to suggest that maybe mining should be done either by non-smokers, or that if smokers were to be permitted in the mines, that a low exposure standard should only be set for those companies who hired the smokers, and much more lenient, or higher exposure standard, for those who hired non-smokers. Fortunately, I think this type of argument did not convince the public officials of the United States to set a dual standard.

It did, however, precipitate a very intense investigation both within and without the government. By 1971 we had demonstrated that there was a significant excess of lung cancer in miners exposed to cumulative doses of 120 working level months up to 350. This meant that any individual exposed at one working level for 12 months per year, within ten years had accumulated enough exposure to lead already to a risk of lung cancer of 3 to 4 times. In 1971 Lundin also demonstrated that when you adjusted for cigarette smoking patterns among the uranium miners, it was only sufficient to account for about a 49% increased risk in the expectation, yet there were still 62 observed deaths versus 11 to be expected or a 464% increase. At that time Lundin hypothesized that if the latent period of radiogenic lung cancer was longer in the non-smokers as compared with the smokers, due to the predominance of promoting effects of cigarette smoke, that it might be too early to expect any problem in the non-smoking proportion of the uranium group. Two more recent reports deal with efforts to test this hypothesis among the U. S. uranium miners, one reported in 1974 at the International Cancer Congress and one by Dr. Archer reported at the New York Academy of Sciences in 1976.

There were approximately 632 non-white Indian uranium miners, and out of this group, as determined by the medical examinations and questionnaires solicited in the period of 1950-51, 1954, 1957, and 1960, fully 437 of those were reported to have never smoked cigarettes. The other clearly obvious pattern for those who did smoke is that they smoked very little. That group, when followed up through 1973, demonstrated eleven deaths due to respiratory-tract cancer, whereas you would have expected only 2.5 to occur. That 2.5 expected, I might comment, is fallaciously high due to the fact that the rates used to generate them were from the Colorado Plateau population, which had a component of non-white groups other than Indian. Studies have shown that the Indian populations have a much lower baseline risk of lung cancer. Among those Indians who had died due to lung cancer four had never smoked cigarettes, two had smoked cigarettes only in the quantity of two or three cigarettes a week, four smoked two to six cigarettes a day, and only one smoked a pack or more. The interesting figures here show that the mean induction latent period for those who had never smoked or smoked very little, that is less than one cigarette a day, was 18.5 years. When we contrast that with the observations among the white uranium miners who died from lung cancer, and who had smoked 20 cigarettes or more a day, the mean induction latent period was 13.7 years for the smoking. Clearly, these results support the hypothesis that non-smoking merely protracted the interval from the onset of mining to the induction of the cancer.

These observations have now been confirmed by Axelson and Sundell in their studies of the Swedish zinc miners, where they have shown an association of radon-daughter exposure and lung cancer in non-smokers, and a longer length of induction period for the non-smokers as opposed to smokers. In 1974 Archer _et_ _al_. demonstrated that, as opposed to matched controls, matched on the basis of age and cigarette smoking, there not only was an excess of small-cell undifferentiated tumors in this group but there was an excess of epidermoid and adenocarcinoma also, but to a lesser degree. More recently Dr. Archer and his group demonstrated an exposure-response relationship for radiation and lung cancer for miners within the various smoking and non-smoking categories, indicating that for each group there is an increased risk associated with radon-daughter exposures that persists in the absence of cigarette smoking. Recently, we have seen some more definitive analysis from Czechoslovakia and these studies have demonstrated a statistically significant increased risk in lung cancer in each group of radon-daughter exposure categories, down to and including 100 to 140 working level months.

Now, what can we say about the problem in uranium miners? We can say that by 1974 we had a sizable increase of lung cancer; we can also say we have a sizable increased risk of accidental deaths. More importantly, we can see now what have been the devastating effects of the very lengthy period of public debate and denial of the risks that have gone on. In the same group studied and observed from 1950 through 1974 we now know that the relative risk of lung cancer is very high. In terms of public health impact, the cumulative risk has increased steadily: 9.2 excess deaths by 1962, 174 deaths by 1974. We have 114 excess lung-cancer deaths that have occurred in the study population of 3,000. We now know that as of 1978 there are at least 35 more lung-cancer deaths and 25 have been diagnosed among those 3000 miners systematically followed up.

Does the problem with mining uranium ores stop at the surface of the mine? Obviously, we don't have all the data but the indications are that it may well not. Our original concerns were restricted to the mining population. However, in 1971 we started looking at what happens to the group who were involved in milling. One significant finding was an increased risk of lymphatic cancer in this group.

Finally, can we say that the problems of uranium as a source of energy cease with milling? I think the evidence is now before us that the answer to that question has to be no. During the late 1960's and the early 1970's it became very widespread knowledge throughout the Colorado plateau areas, that the tailings and waste materials from the milling of uranium ores have been used for landfill for housing developments in the Colorado plateau area. At that time studies had indicated that the radon gases were emanating through the surface foundations of the houses and that the concentrations of radon daughters in some of the buildings were similar to what was being experienced in the underground mining. We now know that in Colorado itself seventeen million tons of uranium mill tailings at inactive and abandoned mill sites are sitting with no regulation. The long-term health implications of this situation remains to be demonstrated, but from the data at hand the implications are not reassuring. In that context I think it is noteworthy that in 1978 the Congress finally passed the Uranium Mill Tailing Radiation Control Act, which designated the Environmental Protection Agency to set standards concerning control of past waste disposal from these mills and earmarked the Department of Energy with responsibility for implementing that. Recently, there has been a timetable set forth in the Federal Register of Wednesday, February 28, 1979, with an announcement by the Nuclear Regulatory Commission, giving the timetable for standards that will be set to control the waste disposal problems associated with past and current milling of uranium.

In the context of the underlying theme of this conference, human costs of energy production, or an assessment of health effects associated with energy technology, I think three points are clear. Due to the uncontrolled dissemination of waste materials, the human cost of energy production is not restricted to and within the exclusive domain of the working population. Due to protracted debate and the long period between identification of health problems and subsequent control of exposures, we have and will continue to have a legacy of health effects in the uranium-mining industry for years and years to come. The third point is that premature speculation, which seems to be prevalent in many studies of occupational health, that the effects obviously have to be due to something other than the industrial setting, in this particular case smoking, has led to further delays in the suggestion that we set adequate standards in the United States.

Two questions are now obviously before us as a society, if I may go back to the original theme that I addressed. With the uranium-miner experience as a model, can we as a society afford the years of debate in assessing the health consequences of future energy technology, while exposures are allowed to take place? Certainly we should not. Second, can our society any longer permit a lack of separation of powers in its government, that is the separation of powers between those who promote a technology and those who regulate it? The answer to that is certainly no, too. I think also that the support of research concerning health effects, for example with radiation, but I suggest in the whole energy technology field, should no longer be in the hands of those who both promote and regulate a particular technology.

DISCUSSION

SESSION I

Dr. Leonard Hamilton, Brookhaven National Laboratory: I would like to ask Dr. Morgan how he would have coped with trying to predict the health effects in miners in the period 1985- 1990-2000, if he was involved in an exercise where one would have to calculate these figures?

Dr. Morgan: That is a good question and I think one has to consider the various health hazards to which miners are exposed. If one starts off by considering coal workers' pneumoconiosis, then there is enough information from the National Coal Board of Great Britain and from Reisner of Steinkohlenbergbauvereins of Germany, to calculate the likelihood of a previously non-dust exposed new miner developing simple pneumoconiosis. And from that, since the attack rate for PMF is known for Category 2 and 3 simple pneumoconiosis, one can predict with a fair degree of accuracy how many men are going to develop PMF in those particular years. Such predictions always assume that the dust measurements made by what used to be the Bureau of Mines and now is MESA are accurate. We now know as a result of a Congressional inquiry that something like 40% of the measurements made between 1970 - 1976 were inaccurate. Clearly there has to be some term of established dust sampling program with valid measurements. The second thing one needs to consider is, of course, industrial bronchitis. I really did not have time to point out in my presentation that the type of dust which is responsible for the induction of industrial bronchitis differs from that which is responsible for the development of coal workers' pneumoconiosis. We know that it is the respirable fraction between 0.5 and about 6 microns which leads to the development of coal workers' pneumoconiosis. The evidence would suggest that it is larger particles, between 5 and 10 microns which are deposited in the dead space and that lead to the development of industrial bronchitis. But industrial bronchitis is nothing like as much of a hazard as is cigarette smoke-induced bronchitis. Nevertheless, one also needs to be sampling different sized particles, or better still the total airborne particulates, to obtain some idea of how to predict the prevalence and effects of industrial bronchitis. So, there are two factors to be considered, both of which are likely to produce occupationally related impairment, namely the size of the particle, and the amount of dust that enters the respiratory tract. The third thing one needs to know something about is the cigarette consumption of the miners who are currently working. If one knows this, I think one can draw the necessary inferences from Fletcher and Peto's work in Britain which shows that about 13 to 15 percent of cigarette smokers will go on to develop airways obstruction.* I forget the exact number who develop severe airways obstruction, but these data can be found in the monograph. Occupational exposure does not enter into this area. Finally one must honestly think about accidents. This is not my bailiwick, but clearly there does need to be some attempt made to limit the number of accidents. Does that give you some idea of how I would go about it?

Dr. Hamilton: Reasonable. There is a further point I would like you to clarify and that is this. How would you go about trying to predict what the effects of this 2 mg per cubic meter standard is going to be? Given the uncertainty that you would have stated about measurements. How does one cope with that?

Dr. Morgan: Quite clearly, one has to have valid dust measurements before you can make any accurate predictions. At the time the Bureau of Mines introduced their dust sampling program there were one or two of us in NIOSH, Dr. Marcus Key and myself, who suggested that the personnel sampler measurements should be supplemented by area sampling. We were not too sanguine concerning the accuracy of the personnel sampler, since this is dependent on the flow rate of the cyclone and several other variables. To us some sort of check seemed desirable. In the absence of valid dust measurements, one can look at the attack rate and the progression rate of CWP over the same period. Alternatively utilizing the dust measurements as far as possible, and from the calculated attack rate and progression index, one can try and calculate how many miners will be affected over the next 30 years. In this regard, when I was at NIOSH we used a group of radiologists for the most part to look at progression rates. The only trouble was that there were some radiologists who read twenty times as much progression as did others, and all read about ten times as much progression as did the British readers. While I have a feeling that the British were under-read, I also have a feeling that epidemiological studies cannot be carried out significantly and properly in a situation where individualistic opinions reign supreme. There has to be some standardization and uniformity among readers. However, radiologists are not always susceptible to standardization.

* The Natural History of Chronic Bronchitis and Emphysema, Oxford University Press, 1976.

Dr. Radford: I would like to ask Dr. Seltzer if he would comment on the accident rate in coal mining. He gave some numbers that sounded fairly horrendous to me in the area of safety and accident rates, as they relate to morbidity and mortality. Would you care to comment, Dr. Seltzer?

Dr. Seltzer: The mortality rate in coal mining, in underground coal mining, has decreased significantly since 1969. The main cause of this decrease has been that the multi-victim disasters which plagued the industry throughout the 19th century have been reduced significantly. What have not been reduced are some of the other causes of accidents. What has not been reduced at all since 1950 is the frequency rate of disabling injuries. The 1969 Act did not address specifically the cause of disabling injuries. Data from the Mine Safety and Health Administration (DOL) in 1977 says that there were almost 15,000 disabling injuries which accounted for, on the average, two months lost time, calendar time. If you take the current fatality rate and the current disabling injuries rates, multiply them by the expected number of miners for 1985 and the year 2000, divided according to surface and underground, you can get the order of numbers I gave you.

I'd like to say something on the health question which is Dr. Morgan's bailiwick and less so mine. To think that coal workers' pneumoconiosis is a disease of the past, I think, is to make a severe error. To say that you have to assume several things. One is that the two milligram respirable dust standard is inherently safe. It is not. That standard (2mg/m3) is based on British research of the 1960's. It is based on a number of statistical manipulations which do not look very sound in retrospect. The probablity curves that were constructed with these data suggested that at two milligrams, one to two percent of the miners exposed would contract CWP (coal workers' pneumoconiosis) over a 35-year period. Follow-up studies released recently say that the probabilities are even higher by one or two percentage points. The 2 mg standard is not risk-free. Yet it is more stringent than any other standard in the world. I think that before you can make the assumption that CWP is a disease of the past you have to confirm the safeness of the current standard. The second assumption that is made by Dr. Morgan and others who think that CWP is a disease of the past, is that every mine section meets the 2 mg standard every day. That is, what the Marx Brothers used to call, horse feathers. Any one who knows anything about coal mining knows that every section does not meet the 2 mg standard every day. There have been several studies that have confirmed this. The most recent study--done at the University of Kentucky-- indicated that 27% of the dust samples surveyed were impossibly low. The study's author, Gerald Sharp said such measurements were either lower than or at the level of dust measurements taken outside the mine! If you have first-hand knowledge of the mining industry, you know that there are various ways that both the company and the miners can falsify dust level measurements. Everyone knows, including MSHA, that the recorded dust measurements are too low, and because of that knowledge, MSHA is now considering different ways of measuring dust. The most important innovation, which the Mine Workers' Union (UMWA) has proposed, is that miners control the dust sampling. My hunch is that if that takes place, you would see more effective dust control than you have in the past.

Dr. Morgan: May I just comment on two portions of that comment. If MESA has changed its name to MSHA or something else then I apologize for being out of date. I don't doubt that by next year it will have yet another name, another set of initials, and another set of inaccurate dust data. I would point out, however, that my figures were not based upon the 1969 data of Jacobsen. Had Dr. Seltzer been in San Francisco two weeks ago at the International Conference on Occupational Lung Disease, he would have heard Mike Jacobsen give follow-up NCB data based upon complete dust sampling up to the end of last year. He would also have heard Dr. Manfred Reisner do the same thing for German coal mines. The relationship between the likelihood of development of CWP and various dust levels as derived from both the NCB and the German data, are very similar, in fact they almost coincide despite the fact that the Germans use a different method of measuring dust, vis. the Tyndalloscope. Both mathematical models have hardly changed since the first interim figures were presented at the European Iron and Steel Community Meeting at Luxemburg in 1972.* The present data that Jacobson and Reisner have described in the San Francisco Meeting are in very close accord with those originally published by the National Coal Board. Moreover, I did not say that coal workers' pneumoconiosis was a disease of the past. What I said was that the development of further CWP would be highly improbable and almost negligible if the U.S. dust measurements conformed to the standard. However, you are absolutely right that dust measurements cannot be relied upon, but that is your problem, sir, not mine.

Dr. George Moore (U.S. Department of Energy in Pittsburgh): A couple of comments. I would say that coal workers' pneumoconiosis is a disease of the past. Secondly, Dr. Seltzer stated that miners' control of the dust hazard could be a more effective way of dust hazard control. At no time should an employee or miner be allowed to control the dust hazard. The experts should control the dust hazard, for example, the people who have been trained in occupational health and industrial hygiene (MSHA. OSHA, etc.). They set the standards and at no

*Conference on Technical Measures of Dust Suppression in the Mines, Luxemburg, 1972.

time should employees be allowed to control the sampling because if they do and eventually they come down with some type of coal workers' pneumoconiosis or eventually black lung, that agency is held liable. In reference to Dr. Wagoner: In the promulgation of the rules for carcinogens, it was reported in the paper about a week ago that you shouldn't have stringent standards for weak carcinogens. In my opinion once a carcinogen, always a carcinogen. There should be one standard for all of them. And a second question, how much political or bureaucratic influence has been brought to bear in handling of the carcinogens? For example, some time ago, Dr. Bingham who heads OSHA and Ray Marshall, the Secretary of Labor, have threatened to quit because brown lung or byssinosis standard was somewhat compromised. This was mainly due to the political implication. So how much political implications have there been in the setting of carcinogen standards? I ask this question because the people who are responsible for the health and safety of the workers should not compromise on an individual's health.

Dr. Wagoner: With reference to your first statement or question, I'm not quite sure which it is. Yes, I strongly support the position of making no distinction between a "weak carcinogen" and a "strong carcinogen." I think it was Marvin Schneiderman (NCI) who said that in terms of public health practice if you make a distinction between "weak" and "strong carcinogens," the "weak" ones will have the greatest public health impact and if we don't do anything they are probably the most pervasive in our society. The second point with regard to the OSHA carcinogen situation and Dr. Bingham's situation, I prefer to refer that question to Dr. Seltzer or Dr. Y. Alarie. I clearly do make that statement on that, though. If we as a society are going to perpetuate a concept of making societal decisions, then those decisions should be made by society with full knowledge of all data and with full disclosure of those decisions out in a forum of society rather than behind closed government doors. The reason there is no discrepancy today, is that the tricks that are being used on the days when the operators wear the samplers are not the same used when MSHA samples are taken. The main point of the trick is called "the penetration game," and since there are no coal mines in New York, I will explain what "penetration game" is. Simply take a narrower cut, a shorter cut into the coal, and the miner or a continuous miner is not exposed to the dust. MSHA has the right to close something down but it does not have the right to start something up. If an operator chooses not to run the machine in the normal way, or chooses not to run the machine at all, MSHA, on the day it did take the sample, cannot dictate to the section working.

On the other point that you raised, sampling is increased with activity whether MSHA does it or whether the operator does it. In an average mine only 3 or 4 samples on a given worker will be taken. You cannot base any estimates of future disease based on 3 or 4 samples a year, which every study that has been done in this area says they have been inaccurate.

Ed Light (Appalachian Research and Defense Fund, Charleston, West Virginia, also on the ORBES Advisory Committee): I have one comment and one question. First, on Dr. Morgan's topic, this is a little outside my area of expertise. There is very strong disagreement with his opinions. Recently I was in touch with Dr. Rasmussen who is with the Appalachian Pulmonary Laboratory in Beckley, West Virginia. Dr. Rasmussen has personally examined hundreds of coal miners disabled with respiratory disease problems. He has given a statement which I am submitting into the record of the meeting. "Emphysema among coal miners is believed to be more frequent than among the general population by some observers and may be a frequent cause of disability among coal miners. The occupational versus non-occupational factors in causing emphysema must be further elucidated. It is true, for example, that cigarette-smoking coal miners, on the average, have more loss of lung function than non-smoking coal miners. On the other hand, there is no proof that cigarette smoking is a greater hazard than exposure to coal mine atmosphere. Some non-smoking coal miners are severely disabled while some heavy smoking miners are healthy. Smoking and moderate exposure may be additive. Much more evaluations will be required to resolve these areas. Strict enforcement of present regulations, careful health monitoring with an effective transfer program with continuing study of the bronchial disease problems offer the best means for preventing the unwarranted suffering and excess mortality among miners employed over the past several decades in the United States."

Now, my question which should be directed to Dr. Wagoner. Is it possible to predict whether there will be additional lung cancer cases from the future production of uranium in the United States , both occupational for the miners and from the waste disposal problem? And second of all, would there be some excess lung cancer cases even if we meet the occupational and EPA standards? Is it possible to make a judgment estimate as to, for example, how many cases of lung cancer we may expect per ton of additional uranium ore needed for power plants?

Dr. Wagoner: I think the current data would indicate two things. Just because we lowered the concentrations, as indicated by the standard, to 0.3 working levels or less than 4 working level months over a 12 month period, does not mean we solved the problem arising because of what we failed to do in the past. That is going to be with us for a long, long period. The second part of that question is--is the current standard that good? Based upon the new data that have come from Czechoslovakia and our own data, my personal opinion is that the scientific data did not demonstrate and support the accuracy of that standard in terms of being protective against future introduction of lung cancer. I think the Czechoslovaks have made some estimate of what they would consider to be the contribution and I would refer the answer, after I get through with this, to Dr. Radford who is probably much more knowledgeable, with his responsibilities of the BEIR Committee. But the Czechoslovaks indicated that they believe that the best measure would be an additional 0.23 lung cancer per thousand miners per working level.

Dr. Radford: I do think that we have a substantially greater body of information on the problem of radon daughters and cancer induction than we had even 3 or 4 years ago, because of a number of new studies including further followup on fluorospar miners in Newfoundland, Canada, and the uranium miners in Canada, which, in my opinion, constitute probably the most significant group in terms of defining the risk of any that I know of. They have not, however, yet been followed up adequately, it is pertinent to ask whether they will be. In addition to the Swedish zinc miners that Dr. Wagoner mentioned, there have been a number of other studies carried out on Swedish iron miners, and I'm doing one of these myself. I think that the Canadian miner data especially indicate that even for exposures down in the range of 30 working level months, there appears to be a doubling of the lung cancer risk. At least my preliminary evaluation of the published Canadian miner data suggests this. Thirty level working months: that means a tenth of a working level for 300 months, which is 25 years exposure to a 0.1 working level, almost doubles the cancer risk. Is that an acceptable standard? I think more information is going to be coming out in the next 3 or 4 years which will enable us to sharpen that, but the point I wanted to add to this discussion, is concerned with what Dr. Wagoner mentioned about the mine tailings issue of uranium fuel sites. We have a problem right here in Pennsylvania which is on the front page of today's paper, about the Cannonsburg, Pennsylvania site. This area has now been found to be heavily contaminated with uranium ore tailings, and indeed if you visit the community, you find out that they may have carried some of these tailings all over the western part of the state, at least according to some anecdotal statements. So, that here we have one such problem right in our own back yard-- it isn't just in Denver, Colorado, Grand Junction, or elsewhere in the uranium mining areas, it's around the U.S. The legislation referred to by Dr. Wagoner named 22 sites in the United States and the list is growing. These are not particlarly related to energy production except that the mine tailings issue in the case of coal and nuclear fuel is one that is going to be around for quite awhile.

Jerry Fields (Pennsylvania Power and Light Co. in Allentown): I have a question for Dr. Wagoner or Dr. Radford. In a recent hearing we had for a nuclear plant that we're building and also other hearings, the U. S. Nuclear Regulatory Commission has prepared a Table S-3, that deals with the nuclear fuel cycle. The question that generally comes up from intervening groups are the effects of milling. My question is of the total fuel cycle how significant is the milling portion of it? Both from the environmental and the cost benefit analysis points of view?

Dr. Radford: Well, my answer would be that from the data we have so far, the occupational health problem in the mills is probably not a major one. If indeed it is a problem then rather than being lung cancer, the data suggest a very different mechanism of action, which will probably have to be controlled by different techniques than eliminating radon daughters. But the milling issue is intimately tied up with environmental effects of the mine tailings. According to EPA (I'm not sure I believe this, but this is what they say) in the whole nuclear fuel cycle the exposures from mine tailings lead to the biggest single health impact.

Dr. Hamilton: I would like to comment on that last point. When you say something is the biggest part of the nuclear fuel cycle it could be biggest because all the rest is terribly small. Our group has done some calculations, in which we relate mining and milling to the exposure that you get. The risk to an individual accumulating a dose to the bronchial epethelium over a 50 year life period-- any individual from now to eternity--developing lung cancer that results from one year's operation is something like 1 in 10, as compared with the risk of developing a lung cancer from natural background, which is something like 10 times greater. It only becomes a significant risk if you take this dose and multiply it, as some people have done, by the world population from now to the next 25 million years and calculate the number of people that would die from lung cancer.

Dr. Radford: I think your point is well taken. You may have noticed that I was careful to say that I'm not sure I agree with this assessment. But, this kind of evaluation is really what the purpose of this symposium is all about. Can we make predictions of this type in comparison with coal workers' pneumoconiosis?

Dr. Halen (Jones and Laughlin Steel Co.): Insofar as accident statistics are concerned, a large number of variables determine whether a man or miner loses time from work and how much time he does lose. I wonder if the statistics that are available today are responsive to the varying severity of accidents as measured by an objective assessment of injury, or do they report only total lost time?

Dr. Seltzer: I'm not sure of all the variables that you're talking about. What I was referring to in the two months figure, was the average lost calendar time of temporary total injuries, not the fatal injuries and not the permanent partial injuries. I'd like to add one other thing. These accident statistics are probably as advanced as any you can find. Informally, I am sure MSHA would admit that in the past, perhaps as much as 60% underreporting and undercounting existed in disabling injuries. With new regulations being promulgated within the past year, MSHA now estimates about 20% underreporting and undercounting. Thus, the available information is not complete and is hampered by underreporting and undercounting. The main variable in that equation is what is called "Light Duty Policy." That is when a person gets injured on the job and is encouraged not to record it. That injury is not reported as a disabling injury. It is reported as a nondisabling injury and that worker is kept on the payroll. There are very substantial benefits, both from the company's point of view and the worker's point of view to play along with that game.

Dr. Halen: Well, let me answer that just for a minute. I think there are ways of our objectively measuring the severity of injuries, if one goes down the line of a computation, fracture and so forth. What I mean, for example, in a recent survey that I myself did prior to a strike, when we had some 100 coal miners off, there were three with fractures and 97 with sprains and strains. I just wondered--is there a way--or do these statistics address this sort of thing so that one can determine more accurately what can be prevented and what cannot be prevented?

Dr. K. Morgan: May I say something? I find myself in agreement with Dr. Seltzer for once. If you consider fatality rates in coal mines a relatively objective finding, the National Academy of Sciences report indicated that the rate in the United States is three times to seven times as high as in most European coal mines. I think you have to distinguish between the accident rates at large companies, such as that of Dr. Halen, which have a good record, and the many small coal mines which employ 15 or 20 miners in West Virginia and Kentucky. They're the ones which have the bad accident record. It's not United States Steel and Consolidation Coal Company, which have made a real effort to cut down accidents, but the small coal mines or the relatively small coal mines, where the record is bad. I don't think there's much doubt that this is where something should and could be done.

Dr. Hugh Spencer: The recent discussion has brought up one more issue that I want to address. The question of radioactivity in the mine tailings of coal mines, has thus far received only a brief comment. For example, in the Western fields, some of the lignites are considered more valuable as a source of low grade uranium ores than as a fossil fuel. In the Eastern fields, coals are traditionally uranium poor, having a very small quantity. But the shale overburdens are somewhat hot. One question I'd like to address here is whether or not we have any kind of statistical details or any knowledge of what the radon daughter effect may be on the present population and on the miner himself. There are some 2 million, perhaps 3 million acres of strip mined, unreclaimed land and tailings containing these shales lying on the surface.

Dr. K. Morgan: First, you'd have to show there is an increased radioactivity in the coal mines and I don't think that there is any evidence that this is so. I don't know about the new lignite mines, but the measurements in established mines have not shown any increased radioactivity. The other point is that the coal miners, as a whole, have a lower death rate from lung cancer than does the general population. Now, coal miners are altogether different from the uranium miners of Ontario or Utah. Somebody needs to make the measurements to see whether there is an increase of radioactivity and I think this has been done. To my knowledge, I don't think that there is any evidence that there is any increased radioactivity.

Dr. Hamilton: There is good evidence that Dr. Morgan's remarks are correct.

Dr. Spencer: Let me advise you that Exxon Company locates seams by dropping a Geiger Counter down through the hole in order to find them. And I am quite amazed at the amount of radioactivity in coal.

Dr. Radford: I would like to follow up on Dr. Morgan's response. The fact of the matter is that measurements have been made in underground coal mines and the radon gas levels are not high, and there is not as a result a large rise in lung cancer rates in underground coal mines. I think Dr. Rockette is here, and I believe he has shown a slight excess, nothing like the kind of excess of lung cancer that is found in uranium mines.

Dr. H. Spencer: I accept that that is quite correct. I have no argument with that.

Dr. Morgan: Is Dr. Rockette here because I think it would be very apropos for him to comment since in his report which he provided to the Public Health Service, he had reported an SMR of 112 and he very cautiously, (and he is often misquoted,) put in a caveat that this slightly increased rate could be due to different smoking habits or different geographical locations. In this regard it subsequently turns out that most of the miners who had an increased death rate from lung cancer came from areas in the United States where lung cancer rate is appreciably higher than elsewhere. In this case it was Charleston, West Virginia, but one only need look at the statistics to see that West Virginia, Kentucky, New Jersey and New York for example, have a much higher death rate from lung cancer as compared to Nebraska and Iowa.

Dr. Spencer: I come from the Johns Hopkins University School of Hygiene and Public Health as a biochemist with a long history of study in the field of molecular biology. And since that time, it has been my conclusion that there is no such thing as a safe dose of this sort of thing. The fact that there is a small concentration only means that the impact may be small as a result of only concentration alone. The pathways are still the same. I think this means that zero dose is probably the only safe dose.

Dr. Howard Rockette, University of Pittsburgh: In response to Dr. Morgan, I think the SMR of 112 speaks for itself in terms of having not been able to take into account the smoking history and geographical differences. In the report I purposely pointed that out. Although the SMR of 112 was statistically significant, statistical significance depends upon the sample size also, as well as the actual difference being observed. I think that on the basis of the study I have done, it would be very difficult to go back, since I did not have smoking histories, and attribute that excess, that 112 rather, to be related to occupation. I might note that in the other studies done in the United States, there are two contradictory sets of results. There is the work by Ortmeyer and Costello which obtained a very low lung cancer rate, and also there were some previous studies done by Enterline which obtained a high lung cancer rate. The current study is kind of intermediate between the two, but as I indicated, without taking into account the smoking histories, I don't see how one could attribute a small excess to occupation.

Dr. Morgan: But Ortmeyer did take into account smoking histories, all of which were available. His lung cancer studies are still low.

Dr. Rockette: Yes, that's true. Although I haven't looked at that paper for some time, one thing that did confuse me was that one age subgroup showed an unusually high risk. It was the 65-69 age group. I'm not saying that this means there is an excess risk but I thought there was a peculiarity in that there would be one age group that would have that high of an excess, and it merits further investigation.

Dr. Ian Higgins, University of Michigan: I want to comment on this. In Britain during the 1930's through 1950's, lung cancer death rates among coal miners were low or very low. At that time coal miners tended to smoke less than the rest of the population. This was probably the reason for their low cancer death rates. Coal miners in Britain are now tending to smoke much more heavily. This change is being reflected in the Registrar General's Decennial Occupational mortality statistics for 1970, which shows their lung cancer death rates to be higher. It will be interesting to see what happens in 1980.

I also want to comment on the relative importance of mining and smoking for bronchitis and disability. Comparisons of the prevalence of respiratory symptoms, bronchitis and level of ventilatory lung function in miners and ex-miners and non-miners living in the same area have consistently shown a higher prevalence of respiratory symptoms and chronic bronchitis among miners. Miners and ex-miners also have a consistently lower average lung function than non-miners. The magnitude of these differences in different places has varied considerably. Sometimes miners have appeared very little worse in either respect from non-miners. Furthermore, it has not been very clearly shown that the miners' excess symptoms and lower lung function are due to their dust exposure, though this of course seems likely.

In contrast, respiratory symptom prevalence and chronic bronchitis are consistently related to smoking. Ventilatory lung function is also lower in cigarette smokers than in non-smokers. Thus both mining and smoking appear to contribute to the excess symptoms and lower lung function of miners. My view is that smoking does so rather more than mining; but I do not think mining can be ignored.

In connection with Dr. Spencer's question, I believe it is important to compare miners with non-miners in the same general area where the miners live. It is unsatisfactory to compare miners in Utah with non-miners in the midwest.

Dr. Hamilton: Not a question, but just a comment to the person who was worried about radioactivity in the coal. I have a ball park figure that the worst could represent 2% of radon exposure from uranium. So it is proportionally a great deal less.

Dr. Wagoner: I would like to make a few comments and I hope Dr. Higgins would address some of these this afternoon. In the whole discussion of the coal studies, the appropriate use of regional versus a rather diluted national comparison needs clarification. Also, I think, it is very important that we define the concepts of the "healthy worker" and the "healthy survivor", and our reliance, and I say this as a statistician, on that magical test of significance which generates SMR of around 100. I don't know what a normal SMR is. Certainly there have been discussions where being normal is 60 and normal is 80. I think also one has to consider with regard to the coal studies that maybe they haven't proven the point that there is a problem, but they certainly have not excluded the problem. It is not a negative study.

SESSION II: SPECIAL METHODOLOGIC PROBLEMS IN DETECTING HEALTH EFFECTS FROM FUEL CYCLE POLLUTANTS

Monday afternoon, March 19, 1979

Moderator: Herschel E. Griffin, M.D., Dean
Graduate School of Public Health
University of Pittsburgh

MEASUREMENT OF OCCUPATIONAL AND ENVIRONMENTAL EXPOSURES
By
Morton Lippmann, Ph.D.

EPIDEMIOLOGICAL STUDIES OF HUMAN HEALTH EFFECTS
By
Ian Higgins, M.D.

ROLE OF ANIMAL EXPERIMENTS IN RELATION TO HUMAN HEALTH EFFECTS
By
Yves Alarie, Ph.D.

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MEASUREMENT OF OCCUPATIONAL AND ENVIRONMENTAL EXPOSURES

By

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MEASUREMENT OF OCCUPATIONAL AND ENVIRONMENTAL EXPOSURES

MORTON LIPPMANN

INTRODUCTION

This session is focussed on the detection of health effects associated with the exposures of people to fuel cycle pollutants. While health effects cannot be determined by measurements of occupational and environmental exposures, such measurements can provide a rational basis for predicting the extent and severity of the health effects resulting from the exposures when the nature of the effects and the dose-response relationships are known. The establishment of causal and temporal relationships between exposures and effects must come from epidemiological studies and animal experiments of the types to be discussed by Drs. Higgins and Alarie in the next two papers.

The adequacy of measurement techniques for occupational and environmental exposure evaluations can only be defined in terms of specific contaminants. Thus, in order to define methodologic problems in the measurement of exposures to fuel cycle pollutants, it is necessary to define which contaminants are likely to be encountered in concentrations sufficient to product effects.

Characterizing Exposures to Fuel Cycle Pollutants

For fuel with well-developed technologies, the exposures of concern have been well characterized and techniques for their evaluation have, for the most part, been established. Thus, while the potential for excessive exposures still exist in fuel oil refining, coal handling, and uranium refining, there are no serious deficiencies in measurement and control technology. The excessive exposures that still occur are due to failures to apply the available technologies.

For the plutonium or thorium fuel cycles, i.e., breeder reactor technologies, there are a number of aspects of the exposure control technologies which require considerably more development. These include capture and disposal of the radioactive noble gas fission products, separation and long-term storage of long-lived liquid and solid activation and fission products, etc. On the other hand, there do not appear to be any major technical deficiencies in the measurement technologies for characterizing exposures to radionuclides or ionizing radiation.

The major methodologic problems in exposure evaluations for fuel cycle pollutants are associated with the developing technologies for producing synthetic liquid and gaseous fuels from coal and oil shale. One difficulty lies in defining the compositions and strengths of the effluents and fugitive emissions. There are complex mixtures of organics at various points along the process streams. Furthermore, the stream composition varies greatly from process to process, and with the compositions of the starting materials and operating parameters in each process. The exposures of the refinery workers will also depend on the number, distribution and magnitude of the process leaks, while the exposures of downwind and downstream populations will depend on the design and performance of the effluent recovery systems and the extent of fugitive emissions.

The only data currently available on exposures resulting from these processes are from pilot-scale operations, and there are very few of these. In any case, the results have limited predictive value for full-scale plants because of likely variations in operating factors, and because the effluents acceptable in a pilot-scale operation are likely to be unacceptable in a full-scale plant. Thus, the controls specified on the basis of the pilot-plant or demonstration plant experience should prevent the release of hazardous effluents into the occupational or community environment of the full-scale plant.

While the exact compositions of the process and waste streams from coal conversion and oil shale processing plants will be highly variable, it is clear that they will contain large numbers of hydrocarbons that are mutagens, teratogens, whole carcinogens, co-carcinogens, initiators, and tumor promotors. Thus, even if the compositions could be precisely determined, and if the health effects of each alone were also known, it still would not be possible to realistically determine the potential for adverse effects of the mixture. The combined effects could easily be greater or less than the aggregate of the effects produced by each of the components when acting alone.

Use of Indicator Compounds

One approach to characterizing the hazard potential of such mixtures is to select one or more individual components of the mixture to serve as indicators. The presumption is that the concentration of the indicator(s) varies directly with the hazard potential of the overall mixture. For gasification plants, carbon monixide (CO) may serve as a suitable indicator for most of the plant population. When the CO levels are monitored and results are used to ensure that the plant is operated in a manner that controls the escape of CO, it can generally be presumed that the concentrations of other air contaminants will also be sufficiently low to satisfy occupational and environmental standards.

It is much more difficult to specify appropriate indicator chemicals for the mixed contaminants which can escape or be released from coal liquification or oil shale processing. Concern for the carcinogenic potential of the mixture is likely to force tighter controls than those needed for the prevention of either acute or chronic chemical toxicity. Unfortunately, there is no single chemical which can serve as an acceptable hazard indicator for this type of process.

Some investigators have adopted or suggested benzo(a)pyrene (BaP) as an indicator chemical, on the basis that: 1) it is likely to be present in most mixtures, 2) it is a known and potent animal carcinogen, and 3) it can be routinely analyzed by analytical techniques of proven reliability. While it is likely to be present to some degree in most mixtures, its contribution by mass or biological activity is likely to be highly variable. Furthermore, its potency as a human carcinogen is uncertain.

An epidemiologic study of roofers exposed to very high concentrations of BaP found some excess lung cancer among those exposed for more than 20 years (1). The SMR's for those exposed for 9-19, 20-29, 30-39, and $\geq$ 40 years were 92, 152, 150 and 247 respectively (the smoking histories of the workers were not known). Airborne BaP concentrations measured in roofing operations range from 14 $\mu g/m^3$ in the roof-tarring area, to 6000 $\mu g/m^3$ in the coal-tar roofing kettle area (1,2). The amounts of BaP recovered from masks, which were worn by a minority of roofers studied, indicated an average of

16.7 μg BaP inhaled per day (1). Another group exposed to high concentrations of airborne BaP were British gasworkers, who inhaled about 30 μg/day (3).

The mainstream smoke of a cigarette contained ∿ 3.5×10^{-2} μg of BaP in 1960, (4) although the levels are lower now. Thus a 2 pack/day smoker inhaled ∿1.4 μg/day. Ambient urban air in 1958 contained about 6 ng/m^3, (2) which could account for an inhaled mass of ∿ 120 ng/day, i.e., 0.12 μg/day. Rural ambient air has about 10% as much BaP(5).

The lung cancer SMR's for rural non-smokers, urban non-smokers, light smokers (≤ 1 pack/day), heavy smokers (> 1 pack/day), roofers (≥ 20 years exposure, and including many smokers) and gasworkers (including smokers) are 14(6), 16(6), 124(6), 502(6), 159(1), and 169(7), while their daily BaP exposures are of the order of 0.012, 0.12, ≤0.70, 0.7 to ∿ 2.5, 17, and 30 respectively. Clearly, BaP exposures are not particularly good indicators of lung cancer risks. All of the exposures cited involve a mixture of organic compounds and other toxicants as well, but the influences of each, and their interactions, cannot be determined.

Since there is potential for exposure to so many such substances in coal and oil shale processing, it is impractical to routinely monitor for more than a few indicator compounds. Furthermore, different indicators or groups of indicators may be needed in different situations. Therefore, research is needed on the unit operations and process streams in these industries, to identify chemical indicators whose biological effects are similar to the mixtures in the expected exposure atmospheres. The strategy recommended by a USDOE sponsored Working Group (8) is to identify a variety of potential indicators (perhaps 6 or 8), identify the places where each is likely to be useful and the kind of monitoring required. They concluded that the state-of-the-art for monitoring each compound by the methods needed should be assessed and, if necessary, that new instruments should be developed. Since the final selection and evaluation of specific indicators cannot be made until a commercial plant is built, it is necessary to be able to choose among a number of indicators for which instrumentation is available. The Working Group also recommended that the instrumentation should probably be non-specific, so that a common line of development will lead to instruments capable of measuring several indicator compounds.

MEASUREMENT TECHNIQUES

Exposures to fuel cycle pollutants can take place via several different pathways, i.e., inhalation, skin absorption or penetration, and ingestion. There can also be exposures to excessive levels of physical agents, i.e., noise, ionizing radiations (x and γ radiation, short λ UV) and non-ionizing radiations (long λ UV, visible light, IR, microwaves). Most of the measurements made to determine actual or potential exposures are made of the exposure environment, e.g., concentrations of chemicals in the air, drinking water, food, etc. These can frequently be supplemented by measurements which determine toxicant accumulations or effects, e.g., measurements of in-vivo burdens, concentrations in body fluids or tissues, and concentrations in excreta such as urine, feces, hair, exhaled air, etc.

In-vivo measurements are practical when they can be made with external detectors, as with radionuclides emitting penetrating radiations which can be detected with solid-state and/or scintillation detectors. Population estimates of accumulated chemicals and radionuclides can be made by measurements of tissue burdens, when appropriate tissue samples can be obtained from human accident victims at autopsy.

Sensitive analytical techniques appropriate for determining the contents of tissues, excreta, air and water samples are generally available, although research and development on techniques for concentrating and/or separating the contaminant from its matrix or sampling medium may be necessary in many cases. There are relatively few cases where a judicious selection of separation techniques and laboratory analytical techniques fail to yield satisfactory analytical capabilities for an environmental analysis of interest. However, there may frequently be cases where the technology is too time-consuming and/or expensive for the particular application.

One major area in which our capacity for environmental assessment is technology-limited is in the evaluation of inhalation exposures to complex mixtures of airborne organics. Another area is direct-reading instrumentation for chemicals which produce effects more associated with their transient peaks rather than their cumulative exposures. The need for direct-reading instrumentation is greatest for chemicals present as aerosols. Great progress has been made in the past decade in the development of continuous direct monitoring devices for specific airborne gases and vapors. By contrast, there are no instruments available for the continuous measurement of the airborne concentrations of specific chemicals present in the atmosphere in aerosols. We cannot, for instance, currently monitor the concentration of sulfuric acid mist. Our inability to do so has contributed to our failure to characterize the roles and effects of the various components of the overall SO_x mixtures generally present in the ambient air.

The only direct-reading aerosol monitors currently available are those which measure some physical parameters of the aerosol, i.e., size distributions, overall number or mass concentrations, and light scatter or extinction.

The adequacy of measurements made to evaluate the potential for health effects in a given situation can be judged by a variety of criteria or combinations of criteria.

These include:

1. Sensitivity, i.e., can the airborne concentrations capable of producing the health effects be measured at an acceptable level of accuracy.

2. Specificity, i.e., can the contaminant of interest be measured in the presence of co-contaminants and background contaminants.

3. Temporal resolution, i.e., can the peak concentrations of the contaminant of interest be determined with a resolution appropriate to the averaging time for the biological effect.

4. Temporal responses, i.e., what is the interval between the time of air sampling and the availability of the concentration determined for the sample. This can vary from instantaneous for direct-reading gas phase sensors to months for laboratory evaluations of field-collected extractive samples.

5. Spatial resolution, i.e., can the measurements be made at locations suitable for exposure evaluations. In other words, can the measurements be made using personal samplers or hand-held samplers or monitors at the breathing zone of the individual whose exposure is being determined.

A complete discussion of measurement techniques for the determination of chemical contaminants attributable to fuel cycle pollutants, and for those released into the occupational environment, ambient air, and liquid effluents would require a monograph, and are clearly beyond the scope of this paper. A recent review of the current status of the available measurement techniques and some recommendations for research on environmental measurements was prepared by the Second Task Force for Research Planning in Environmental Health Science (9).

The balance of this paper will consist of a review of techniques for characterizing exposure atmospheres which are applicable to both ambient air and workroom air. Within it, the emphasis is on recent developments in direct-reading instrumentation.

Monitoring Exposure Atmospheres

There are various approaches to monitoring the exposure atmosphere and characterizing the concentrations of the chemicals to which people are exposed. The traditional and simplest monitoring technique is manual sampling, where a known volume of air is drawn at a known flow rate through a collector. For particles, the collector can be a filter; for soluble gases, it can be a bubbler; for organics, it can be a charcoal trap. The appropriate collector is used to trap particles with known, and presumably, high efficiency.

For gases and vapors, the collection efficiency needs to be known and constant. If it is less than 100 percent, the amount collected can be corrected to compensate for the lack of total collection. For aerosols, on the other hand, there must be 100 percent collection efficiency, since the collection efficiency is particle size-dependent, and the investigator wants to know the total amount, and not some estimate that varies with its particle size distribution. In practice, this requirement doesn't cause any great problem, since filters and other types of collectors with essentially quantitative collection capabilities are readily available.

One advantage of sample collection and subsequent analysis is that there is almost no limit in the choice of sample processing that can be done, or in the range of sophisticated and sensitive laboratory instruments that can be brought to bear on analytical problems. The investigator can get the ultimate in sensitivity and specificity, with correction for interferences. On the other hand, there is a basic limitation in this procedure in that there is a significant time lag between sample collection and the determination of what was collected. Manual sampling techniques may, therefore, be used to back up continuous monitors, and perhaps, as the final arbiter on a substance's exact concentration.

For a more complete discussion of the state-of-the-art of air sampling technology and for descriptions of available sampling and monitoring instrumentation, the reader is referred to the latest edition of the ACGIH reference book: "Air Sampling Instruments."(10).

Intermittent/Continuous Instrumentation

There are two basic types of automated instrumentation. One is the intermittent type of operation, and the other is continuous. For monitoring particle size distribution, a combination of intermittent techniques is generally used because there is a very large particle size range to be covered, and there is no universal instrument that can indicate the concentration of each particle size interval over the entire range.

One of the instruments used to measure very small particles (0.08 μm to 0.5 μm) is the electrical aerosol analyzer shown in Figure 1. It sorts out the particles of a poly-dispersed aerosol according to their electrical mobility. The particles are collected on a current collecting filter at the end of the tube, and the amount of charge they deposit on that filter is measured incrementally. The voltage gradient and/or the flow rate is changed sequentially to allow a different size-cut to reach the filter. In this way, there is a progression of size increments. This technique can provide an accurate size distribution in a stable atmosphere. However, it takes a finite amount of time to run through these increments and collect the size band data to get the size analysis.

While the mobility analyzer can be used as a size analyzer for small particles, it cannot be used to measure particles larger than approximately 0.5 μm in diameter. Fortunately, there are other techniques suitable for measuring the size distribution of the larger particles.

The property of light scatter is used in measuring the concentrations and size distributions of particles of 0.3 μm diameter and larger. When light is focused on a particle in the instrument illustrated in Figure 2, some of that light will be scattered. The amount of light that will be scattered by a given particle depends on its size. A photomultiplier can be used to detect the light output from each particle. The pulses can be accumulated according to size intervals in a multichannel pulse-height analyzer, and these intervals can be calibrated according to particle size. Unfortunately, there are other properties of the particle besides its size that affect the amount of light scattered, including the refractive index, the color, the shape, and so forth, and reliable data depend on accurate calibrations.

Intermittent techniques can also be used in monitoring gases. One method for carbon monoxide involves a combination of gas chromatography and flame ionization detection. The flame ionization detector is nonspecific, but by coupling it with the holdup time in the chromatographic column, specific analyses can be obtained since the carbon monoxide will pass through the column and move into the flame ionization detector at a characteristic time.

The alternative approach, i.e., continuous monitoring, is more commonly used for gases. For example, carbon monoxide can be monitored using an infrared analytical technique which does not collect the sample at all. As shown in Figure 3, the gas is directed through the sample tube. Infrared radiation also passes through the tube at wavelengths that are sensitively absorbed by carbon monoxide. There will be an attenuation of the infrared because of the absorption of the carbon monoxide in the sample tube, and thus, the energy received on the detector will vary with the substance concentration. Operationally, this technique is best implemented by using a reference tube of clean air, and getting the difference in attenuation between clean air and the air containing the carbon monoxide.

Other gaseous constituents absorb infrared energy; for example, water vapor. However, these gases and vapors are wavelength-specific, so by tuning to the wavelength at which carbon monoxide has preferential absorptive capacity, the effect of interference can be removed, giving a sensitive, accurate, and specific analysis of carbon monoxide. The only time lag is the insignificant amount of time it takes to flush the sample through the tube. This, then, is a commonly used method of monitoring carbon monoxide on a continuous basis. The sensitivity is a function of the length of the sample tube, which can be 10 or 20 meters with folded tubes. Similar instruments can also be used in measuring other gases by choosing appropriate wavelengths free of significant absorption interferences.

The flame photometric detector illustrated in Figure 4 is commonly used in measuring the concentrations of sulfur-containing gases. If a sulfur-bearing material is passed through a hydrogen burner, a light photon emission will result that can be detected with sensitive photomultipliers. It is a nonspecific technique in that it gives roughly an equivalent response for hydrogen sulfide, sulfur dioxide and some of the mercaptans. If the only sulfur gas present is sulfur dioxide, then the test is quite specific, but if there are other sulfur gases, the test cannot be specific unless the sulfur gas detector is equipped with a chromatographic column that will feed the sulfur species into the flame-photometer in sequence. Such a composite instrument is, of course, an intermittent sampler.

In most situations, it is desirable to have a built-in calibration device on the concentration monitor to ensure that the concentration indicated on the output chart or on the dial is correct. One of the more common devices used for in-line continuous calibration is the permeation tube. For the flame-photometric detector, the calibration device shown in Figure 4 contains liquid sulfur dioxide sealed into a Teflon tube. The tube is slightly porous to the saturated sulfur dioxide vapor above the liquid in the tube. The rate of permeation of sulfur dioxide vapor out of the tube is very much dependent on the temperature, but is constant at a given temperature. When the permeation tube is held within a constant temperature bath, a known emission rate can be obtained. With an accurately calibrated dilution air flow, the concentration of the calibration gas can be determined, and it can be directed periodically into the analyzer to get a span signal. This is a very convenient method of instrument calibration. There are similar calibration tubes available for nitrogen dioxide, and for a number of hydrocarbons that are readily condensed into liquids at room temperature.

Instruments that measure the light emitted during gas phase reactions of nitric oxide and ozone are also used in monitoring contaminant concentrations in occupational and ambient atmospheres. If an excess of ozone is mixed with the sample containing nitric oxide, as in Figure 5, the amount of light is proportionate to the amount of nitric oxide. This technique can be used for measuring nitrogen dioxide by passing the sampled air through a chemical converter that converts the nitric dioxide to nitric oxide, which can then be measured on a mole for mole basis. Nitric oxide and nitrogen dioxide can be differentiated by sequential readings with and without the converter in-line.

The same basic type of instrument can be used as an ozone monitor by feeding an excess of nitric oxide into the reaction chamber. Other ozone monitors are based on other chemiluminescent reactions of ozone, specifically, with ethylene and organic dyes.

Measuring Aerosol Mass Concentration

Both of the methods described for measuring particle concentrations and size distributions, i.e., the small particle mobility analyzer and the larger particle light-scatter analyzer, measure the diameters of particles and sort them by so many numbers of particles in each size interval. Number concentration is important in many studies, but in studying effects on people or animals, the investigator generally needs to know the aerosol's mass concentration. Since the mass of a particle varies with the cube of the diameter and with the density, the investigator needs to know more than number distribution. On the other hand, the automatic machines accumulate a very good statistical base. Adequate approximations of mass concentrations can sometimes be made by determining the volume distributions from the transformed number distributions. If the particles' density is determined, then a mass median diameter distribution or a concentration can be calculated.

While such data transformations may be justifiable in some cases, the property that is being reported is not being directly measured. It is, therefore, sometimes necessary to determine the aerodynamic size distribution, since this is the distribution of sizes that affects deposition in the respiratory tract. If a system of somewhat redundant measurements can be justified, it is best to use a variety of complementary aerosol measurements involving light scatter, mobility, and aerodynamic properties.

The beta attenuation technique combined with a two-stage collection system, as illustrated in Figure 6, sorts the particles by their aerodynamic sizes and measures the mass in each fraction. Using this technique, the air enters an impaction jet at the top, and particles below its aerodynamic cutoff size will be collected on the back-up filter. The mass of accumulated particles collected at each state can continuously be monitored using the beta attenuation technique. A carbon-14 source is used as a beta emitter. The amount of β-radiation that reaches the detector depends on the β-absorption in the accumulated sample between the source and the detector.

There is only about a 10 percent variation in beta attenuation with mass number (Z), with the exception of hydrogen, and hydrogen does not usually contribute much to aerosol mass. Thus, the amount of beta attenuation by the impacted particles on the first stage indicates how much mass of material has been collected in particles above the impactor's cutoff size, and the attenuation of the particles on the second stage indicates the mass of small particles below the cutoff size. However, the response time of this technique is not very rapid.

Another type of direct monitor of aerosol mass concentration utilizes quartz-crystal oscillators as mass balances, and is illustrated in Figure 7. Very small sample masses can be detected as they accumulate on the quartz crystal oscillator. The quartz crystal is cut into a particular mode, and electrodes are attached on each side. When a high frequency signal is applied, the crystal oscillates, and its oscillation frequency depends on the mass of the crystal.

When particles are deposited on the crystal, its mass increases and the oscillation frequency decreases, therefore, the sample accumulation can be measured by the change in the frequency of the oscillator. In modern instrumentation, frequency counting is relatively simple and precise, and very sensitive. Thus, infinitesimal masses produce readily measureable signals.

However, this technique has its limitations. The sensitive zone does not have a uniform sensitivity. It is greatest at the center of the electrode and falls off toward the periphery. However, it does provide a sensitive indication of mass, and with enough calibration to be sure of its performance under the given operating conditions, can be used as a sensitive mass monitor.

Another instrument that can be used to provide an approximation of mass concentration of particles in the one-tenth to one micron range is the integrating nephelometer, which is illustrated in Figure 8. This technique measures the total scatter of an aerosol. The instrument discussed earlier measures the scatter from individual particles. The nephelometer, by contrast, measures the scatter of a cloud, i.e., the total scatter of all of the particles in the sensing zone.

SUMMARY AND CONCLUSIONS

Existing measurement techniques for the evaluation of occupational and environmental exposures to fuel cycle pollutants are generally adequate for the more fully developed fuel cycles. These include the preparation of coal and petroleum products for combustion, and uranium for nuclear fission. Further improvements in measurement techniques for evaluating exposures to the pollutants for these fuel cycles will be primarily concerned with refinements in instrument convenience, performance and reliability. In many cases, there is room for improvement with respect to size, portability, cost, response time, sensitivity and specificity.

Entirely new measurement technologies need to be developed and refined for some of the pollutants associated with fuel cycles currently under active development, especially coal conversion and oil shale processing. One major problem is that the mixtures of hydrocarbons within these processes and in their effluents are so varied and complex that it is not possible to characterize all of their individual and combined toxicities. A second problem is that designs of commercial scale plants and their potential for effluent and fugitive emissions cannot be determined at this time.

An attractive approach for effective hazard evaluations in the synthetic fuels industry is to select one or more appropriate constituents of the mixtures to serve as indicators. However, considerable research will be needed to serve as a basis for establishing the feasibility and utility of this approach for these processes.

Continuous, direct reading instrumentation for monitoring airborne concentrations of many of the pollutant gases of interest are available. Many of these instruments utilize sensors which respond to gas-phase reaction products and/or spectral absorptions. Direct reading aerosol monitors are available for the measurement of mass concentration and particle size distribution. However, there are no direct reading instruments for measuring the concentrations of specific chemicals present in aerosols. The development of such instruments, while admittedly difficult, represents an area for productive research.

ACKNOWLEDGEMENT

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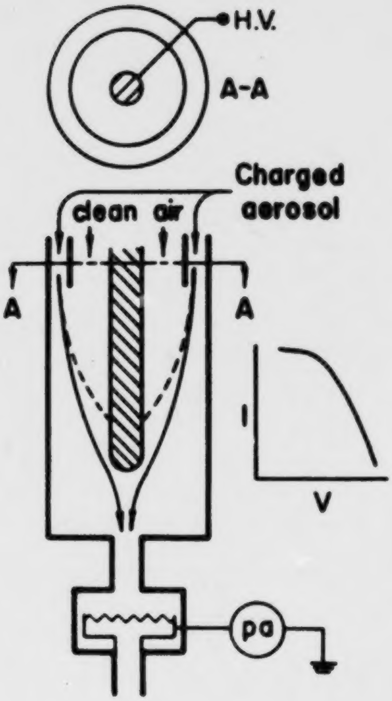

Figure 1. Schematic diagram of electric mobility analyzer tube as used in mobility size analyzer. Source: Liu, B.Y.H., (Ed.), Fine Particles, Academic Press, 1976, p. 599.

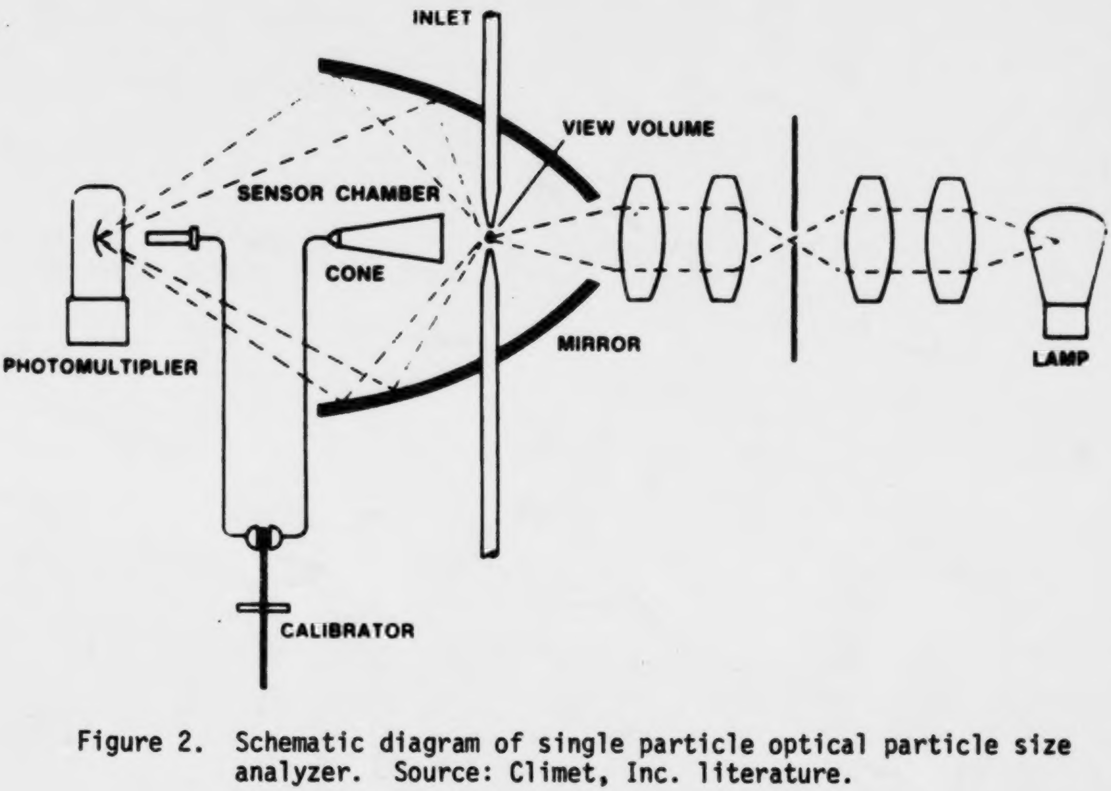

Figure 2. Schematic diagram of single particle optical particle size analyzer. Source: Climet, Inc. literature.

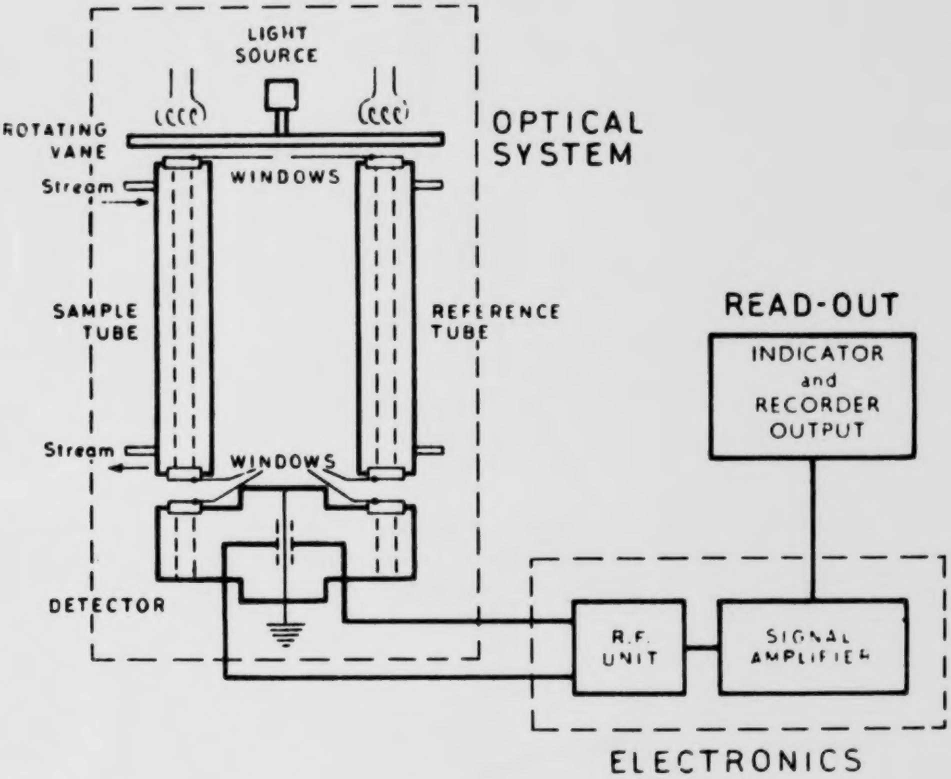

Figure 3. Schematic diagram of infra-red gas analyzer. Source: Air Sampling Instruments (5th Edition), ACGIH, Cincinnati, Ohio, 1978.

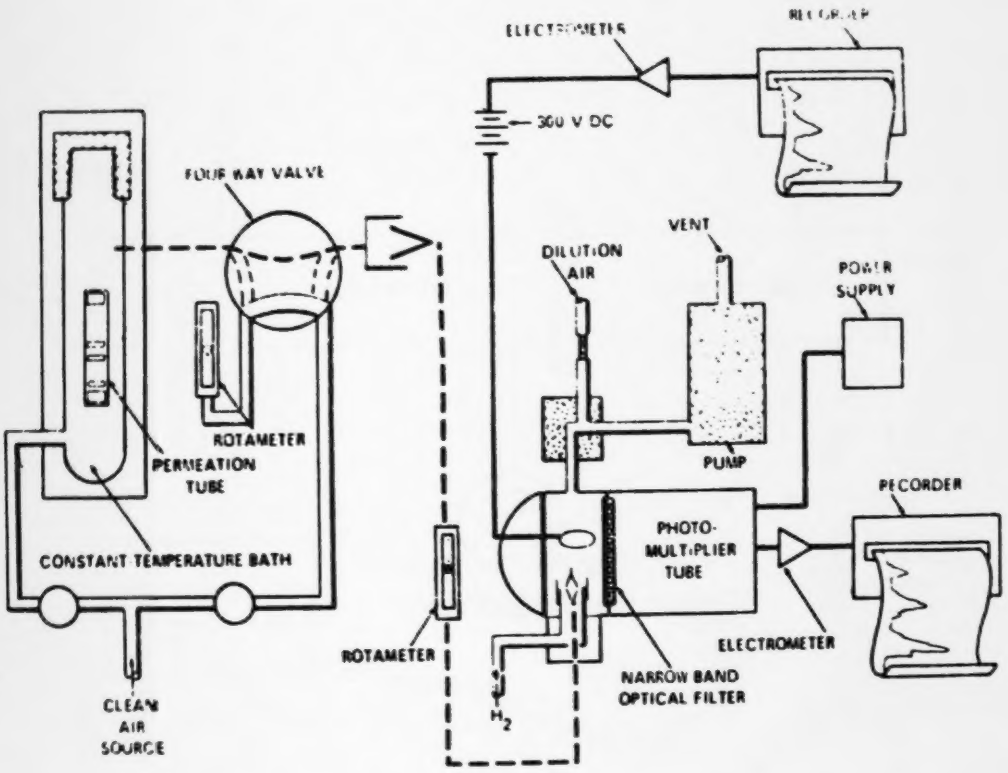

Figure 4. Schematic diagram of flame photometric analyzer for sulfur gases, with permeation tube calibrator. Source: Air Sampling Instruments (5th Edition), ACGIH, Cincinnati, Ohio, 1978.

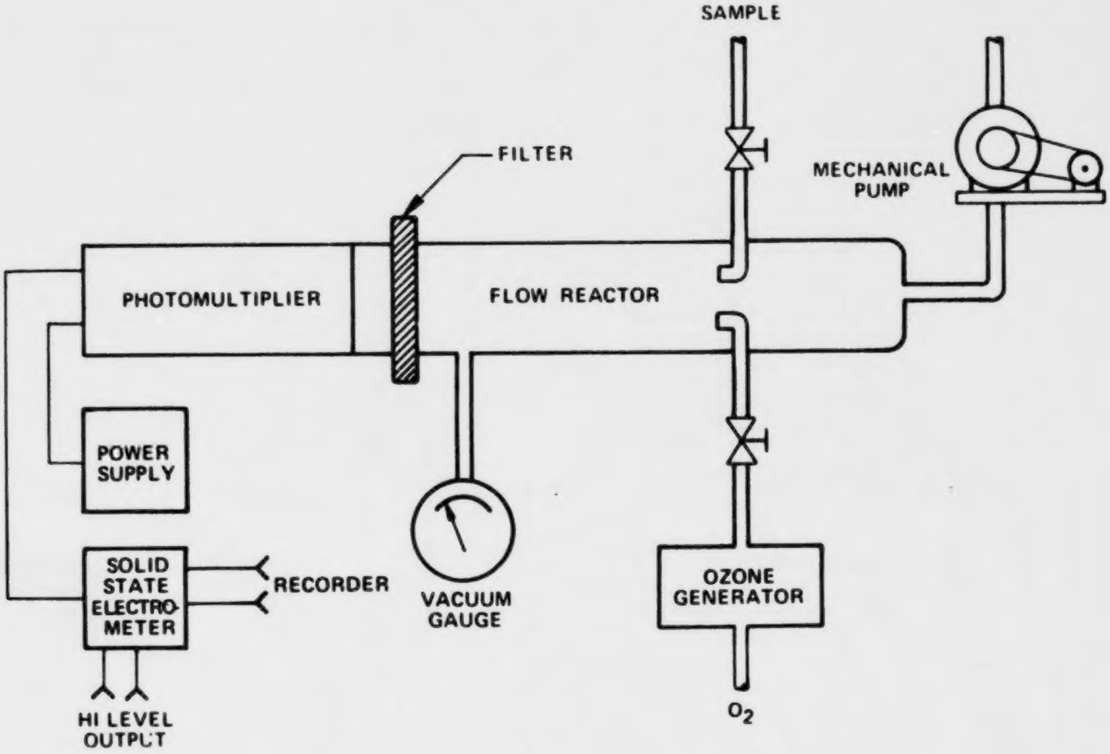

Figure 5. Schematic diagram of chemiluminescence NO/NO_x analyzer. Source: Scott Research Labs literature.

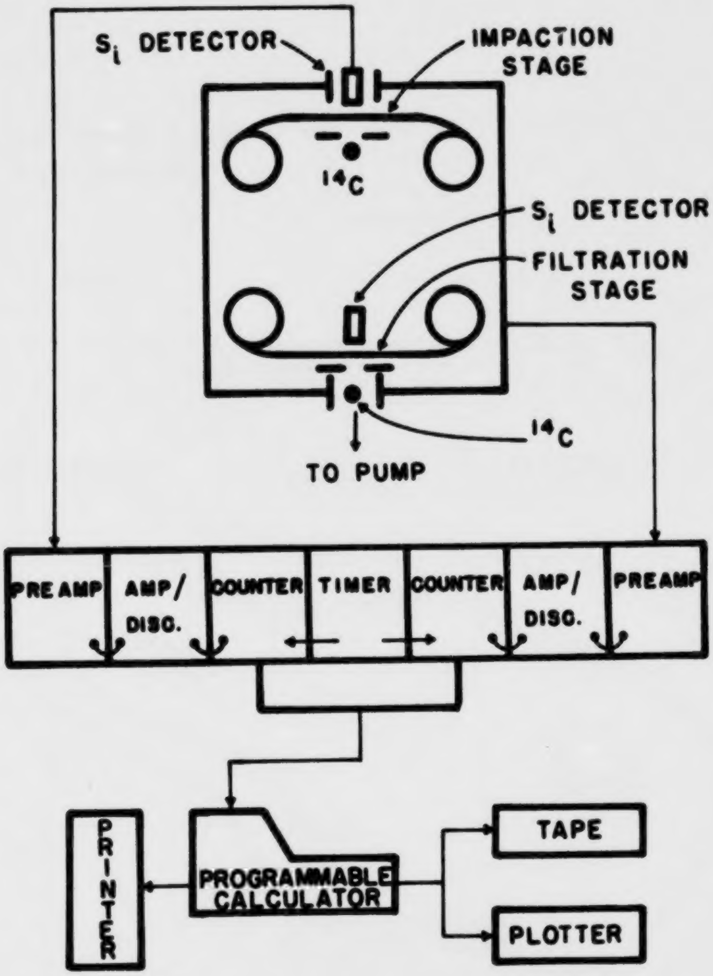

Figure 6. Schematic diagram of TWOMASS mass concentration analyzer. Source: Liu, B.Y.H., (Ed.), Fine Particles, Academic Press, 1976, p. 551.

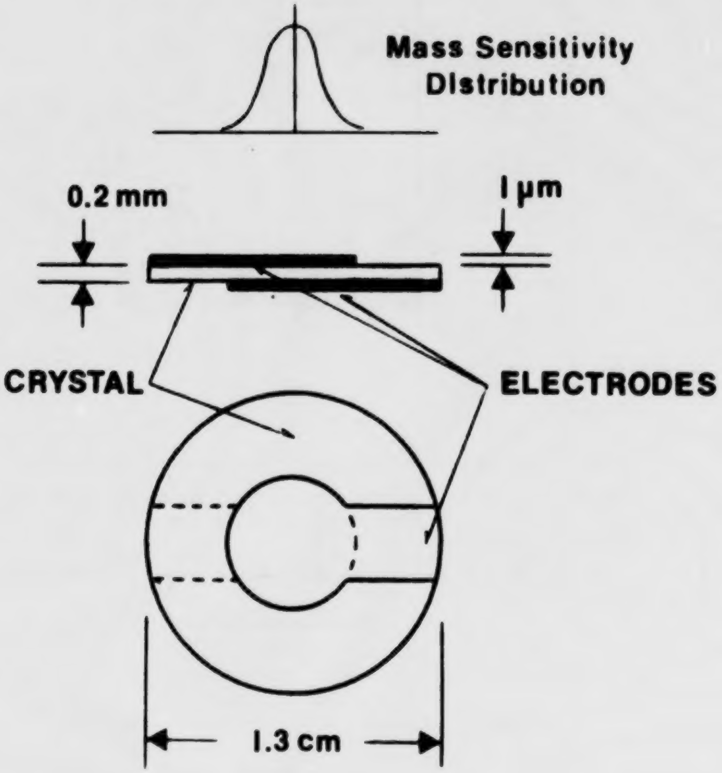

Figure 7. Schematic diagram of quartz-crystal oscillator used as a mass balance, showing sensitivity over crystal face. Source: Liu, B.Y.H., (Ed.), Fine Particles, Academic Press, 1976, p. 489.

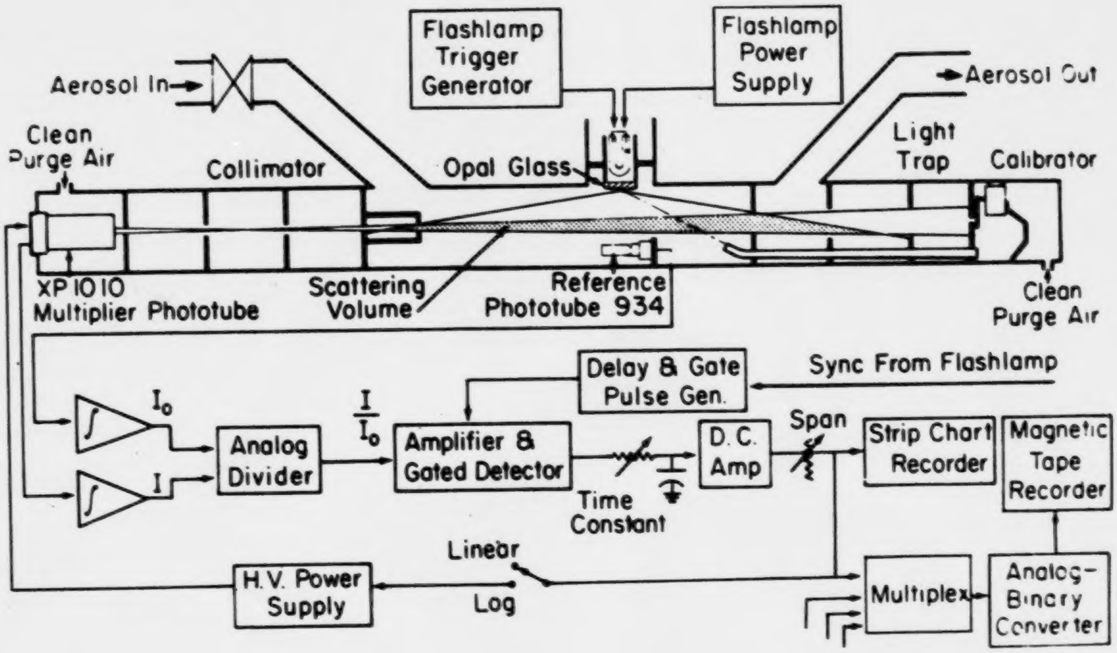

Figure 8. Schematic diagrams of integrating nephelometer.
Source: MRI, Inc. literature.

EPIDEMIOLOGICAL STUDIES OF HUMAN HEALTH EFFECTS

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Introduction.

The task of the epidemiologist who is interested in the effects on health of fuel cycle pollutants is to relate exposure to response in some definable population. As you have just heard, he has first to measure exposure to specified pollutants. He has to do this in the work place and elsewhere to reach sound conclusions about the dosage received by each person in his study group. He has then to identify and measure any effects, or responses, that these exposures have caused. The relationship between exposure and response should be quantified with as much precision as possible in the form of a dose/response curve (or series of dose/response curves). Responses of interest may be influenced by many factors other than pollution, by age, sex, socio-economic status and smoking, for example. It is essential that due allowance should be made for such confounding factors if erroneous conclusions about the hazards of pollution are to be avoided.

As in any scientific endeavor, the epidemiologist can make observations or conduct experiments. Broadly speaking, observations are of two kinds:

1) Persons with an effect of interest can be compared with persons without it to see if their current and past exposure differs, and

2) Persons with known (or estimated) exposures are followed to see if the frequency and severity of some effect can be related to the intensity and duration of those exposures.

Experiments are also of two kinds:

1) Those based on "natural" changes, such as varying concentrations arising from industrial emissions or accidents, and

2) Those based on planned change where the effect of control measures is studied.

Definition of the population for study.

The first problem is to decide what population we should study. Are we more interested in the general population or should we focus on those we suspect of being a high risk group? Patients with heart or lung diseases, asthmatics, the young or the old, for example. Ideally, we should know what proportion of the whole community the high risk group comprises. But susceptible persons have seldom been identified in this way. More often, patients on the lists of physicians have been recruited for diary, panel or other special risk studies. The result has been that one has only a vague idea of the prevalence of such persons in the community.

Instead of trying to study the whole community, which is a taxing activity requiring an initial census, epidemiologists often select some group (or groups) within the whole, which is assumed to reflect the behavior of the whole reasonably accurately. Thus, persons employed in different jobs, children in school, insured persons and so on are often selected as being more readily accessible than members of the community at large. Occupational groups sometimes offer advantages over community defined groups. The late Professor Donald Reid originally conducted his studies of air pollution among postmen (mailmen) because the occupation ensured a certain socio-economic homogeneity.

It may be more practicable to study the effect of certain exposures within a particular industrial group rather than in the general population. Exposure to pollutants, which may occur when coal is converted into liquid or gaseous fuel, and exposure to ionizing radiation are examples. There are a number of problems with occupational groups. The disabled tend to be under-represented. The heavier the exertion required, the less likely are the disabled to remain in the work force. Disabled persons may move from heavier to lighter jobs. Persons with certain impairments and diseases do not ever get into the work force either because they do not try to do so or because they are rejected by a pre-employment physical examination. These forms of selection result in most occupational groups being healthier than the general community, a situation which is often referred to as "the healthy worker effect."

Identification of comparison groups.

It is important to establish adequate comparison groups. Frequently the population being studied can be subdivided into a number of exposure categories (none, light, moderate, heavy; or, better, classes based on pollutant concentrations or dosages). These internal comparisons form the bases for exposure/response curves. In many studies, however, additional external comparisons are important. This is particularly so when the question posed refers to a possibly increased risk of some disease resulting from occupational exposure. Does this group of men who are occupationally exposed to this or that pollutant have an increased risk of dying of cancer? Chronic respiratory disease? All causes? These questions imply some standard of comparison or basis for expectation. The United States population, the population of the state or city where the industry is located, some other industry, all persons covered by social security, are some of the comparison groups which have been suggested and used.

Sources of information on population.

Information on the general population is available from routine demographic and vital statistics. Often, however, the detail one might like is insufficient for adequate comparisons. Occupational data is limited and smoking habits usually nonexistent. If these important confounding variables are to be dealt with adequately, a special effort will have to be made to collect them. Usually an up-to-date definition of the population will need a census.

Occupational groups can be defined by company or Union records. But these may not always be available. They need to be checked for completeness. This can be done through quarterly social security records. Any discrepancies between nominal lists and SSA records need to be resolved.

Measurement of exposure.

We need to decide first what pollutants to measure, how, where and for how long. Often measurements will have been made at one or more sites. But these will seldom be adequate for epidemiological needs. Nevertheless, such measurements may be all that are available for estimation of past exposures. The studies of Buechley[1] and Schimmel[2] and their colleagues on daily mortality in relation to daily pollution in New York City have been based on measurements of pollution made at a single sampling station in uptown Manhattan. Clearly such limited data, while it may provide a reasonable picture of overall change, is less than ideal. In the industrial setting, measurements will usually have been made at sites where the risk of exposure is particularly high.

In neither the general community nor the work place are people exposed to these measured concentrations. In the community, people live at some distance from the sampling station. They work somewhere else. They travel between the two and move about elsewhere during the course of the day.

More important perhaps, but increasingly recognized, they spend most of their time indoors whereas pollution is measured out-of-doors. There are large differences in pollutant levels indoors and outdoors. In the work place employees may spend only a small fraction of the work shift exposed to the pollutant concentrations which are measured. The rest of the time they may be exposed to lower concentrations. An additional problem in assessing exposure at work is that sampling is usually intermittent. The days on which samples are taken may not be representative of all days.

In the general community samples are usually taken over 24 hours, though continuous recording instruments are used for some pollutants. Personal monitors provide an integrated exposure over a working shift or in the case of a radiation badge over a longer period, usually a week. For many exposures we may be more concerned with short peaks than with integrated shift or 24 hour samples. Thus, deterioration in the respiratory condition of patients with chronic bronchitis and emphysema or attacks of asthma may depend more on transient short peaks of exposure than on 24 hour concentrations.

In assessing exposure, the epidemiologist has to be concerned with daily patterns of movement. In the industrial setting this involves a time and motion study. But in the general community this is difficult. Perhaps personal monitoring provides the best way of obtaining reasonably accurate estimates. To date, few such studies have been carried out.

If it is hard to estimate short (24 hour) exposures, it is of course much harder to estimate exposures over the past 10 or 20 years or over a lifetime. The difficulty is compounded by the fact that pollutant concentrations have nearly always changed over the years. What we measure now may have little relevance to pollution in the past. Inevitably we must rely on historical information, work and residential histories and what is known or believed about the way pollution has changed.

Measurement of response.

Turning now to the response side of the equation. A variety of biological indices can be used to study effects of exposure. These range from death, severe disability and serious illness, through respiratory or other symptoms and lung function to mild discomfort, unnoticed changes in physiological or psychological function, or silent accumulation of chemicals in the body. Some of these indices are routinely collected. For example, deaths, including daily deaths, can be obtained from the National Center for Health Statistics (or similar Offices of Vital Statistics in certain other countries). Illnesses are available in insured populations. But many of these indices must be obtained by the epidemiologist by means of a survey.

Death is a good epidemiological index, if somewhat terminal. Provided the fact of death is confirmed by a death certificate there is not much doubt about it. As soon as interest turns to specific causes of death, a number of problems arise. Some of these are concerned with diagnosis. They include such things as criteria for diagnosis, fashions in certification, observer variation and error. Others reflect the ordering of potentially killing illnesses on the death certificate into underlying and contributory causes. Finally, people who die of heart disease do not remain in the population to die of cancer or stroke. There is, as we say, a problem of "competing causes of death."

When one collects ones own data, a clear statement of the biological index used as a measure of the effect of pollution needs to be made. The index needs to be clearly defined and its degree or severity classified. The reproducibility of symptoms, physical signs, special investigations such as chest x-rays, lung function tests, blood and other laboratory tests has to be carefully assessed. Needless to say, initial training is important in ensuring that tests are correctly carried out and variation between observers is reduced to a minimum.

A claim that is sometimes made for epidemiology is that it offers an opportunity to identify persons with the earliest deviations from the norm. While this is certainly true, it should be recognized that minimal changes are often hard to differentiate from normal variation. Once defined there may also be uncertainty about the future implications of such deviations. For example, a modest rise in respiratory airways resistance (50%) may be brought about by exposure to SO_2 or NO_2 at low concentrations. A comparable rise can also occur from breathing cold air, after coughing or even from taking a deep breath. In the case of SO_2, the A.W.R. may revert to normal even while the subject continues to breath SO_2. Do these changes have any more serious implications? We do not know. Yet there is a tendency to assume they do and to regulate accordingly.

Again, in the search for sensitive indices of effects, subtle changes in lung function have sometimes been used. An increased response to carbachol, for example, has been shown to be brought about by prior exposure to low concentrations of NO_2. What such a response means is at present uncertain. But in the present state of our knowledge, such a test seems to me to be an inadequate basis for regulation.

What should we measure?

The brunt of fuel cycle pollutants falls on the respiratory system. It seems obvious, therefore, that our focus of interest should be on this system, though not of course exclusively. We have fairly adequate and reasonably extensive tested methods of investigating the respiratory system. Thus respiratory symptom questionnaires, spirometry, chest radiography and measurement of sputum volume and quality have been extensively used. Recommendations have recently been made by a task group of the American Thoracic Society (ATS 1978). The search goes on for more and more sensitive tests; but in my view, our problem is much more to apply those tests we already have than to develop new ones.

Ionizing radiation and coal conversion products pose a potential risk of cancer, respiratory and other. Collection of morbidity and mortality statistics on cancer should presumably be one of the indices with which we should be concerned. But relatively large populations must be studied if sufficient cases are to develop in a reasonably short time. It is clearly desirable to monitor other changes, which may occur more frequently than cancer, changes in sputum cytology, fetal wastage and congenital malformations, for example, particularly if women are exposed.

The Medical Research Council's Clinical and Population Genetics Unit has been monitoring lymphocyte cultures in 197 dockyard workers exposed to low concentrations of ionizing radiation (cumulative dosages of from 1 to 30 rems).[3] They observed a striking correlation between dose in rem and aberration counts/1000 cells tested. The relationship was particularly notable for cells with unstable aberrations (acentric elements, dicentrics, rings). This suggests to me that lymphocyte cultures should be considered in radiation effects. As other bio-assay systems are identified, consideration should be given to them as they can provide a relatively inexpensive method of screening for harmful effects.

Equal treatment of study population and comparison group.

When an industrial population is being compared with the national, state or other comparison group, it is of course important to avoid introducing bias through greater attention to the group of greater interest. There is a serious risk that this may happen. There is an almost irresistable tendency to correct deaths which one knows to have been wrongly certified in the group of special interest, as a result of review of the hospital records, for example. Clearly such "correction" is quite improper unless a similar scrutiny of the deaths in the comparison population is also made. A more subtle form of this bias is to increase the autopsy rates in the population of interest (but not in the comparison group).

There may even be problems resulting from the scrutiny and coding of the death certificates in the population of interest but accepting the original coding for the comparison group. This seems to me to be particularly likely to occur when the original coding would have been carried out under a different revision of the international classification of causes of death.

Confounding factors.

Many factors other than air pollution influence morbidity and mortality from the respiratory diseases. Smoking, occupational exposures, infections, allergy, heredity, age and sex must be carefully considered before respiratory disease is attributed to general air pollution. Climate, in particular temperature level and short-term temperature changes, may also contribute significantly to disease, both respiratory and other. There may also be difficulty in separating one pollutant from others. In London, for example, daily concentrations of smoke and SO_2 are so highly correlated that it is impossible to separate their independent contributions to effects. In most cities, poor people tend to live in areas where pollution is highest. Inevitably, effects of pollution are therefore confounded with effects due to low socio-economic status.

Many studies of air pollution have failed to allow at all for smoking, occupation, ethnic origin and socio-economic status, making any conclusions that are drawn about the effects of pollution uncertain. Other studies have made some allowance for some of these factors but this allowance may not have been sufficient to eliminate the confounding effect.

Miscellaneous problems.

A variety of problems arise in studying effects in relation to short-term changes in pollution. Studies using daily deaths have shown that the date a death is registered may not be that on which the death occurred (Sunday deaths may be registered on Monday and other day-of-the-week effects may also occur). Place of death may not be that reported. Pollution in most great cities varies considerably from place to place. Attempts to improve dose estimates by considering areas within the city lead to problems with small numbers and uncertainty with respect to how the population at risk of dying may have varied throughout the 24 hours.

Another problem with daily studies is that there is often a phase shift between the measurements of pollution and the measurements of the health effects. Pollution over the 24 hours may be measured from 8:00 a.m. to 8:00 a.m. Whereas, health may be measured from midnight to midnight.

It is difficult to make personal observations or tests for many days at a time. We tried to study a sample of elderly persons (over 65 years of age) in Tampa, measuring their ventilatory lung function once a day. The best we were able to achieve was to follow a small, declining sample of persons for three two-week periods. Over six months diaries or telephone interviews may be more successful, but these suffer from subjectivity and the problem of remembering to fill in the daily record. Another problem is the tendency of most large cities to announce their daily "murk" index on the radio or television, with the possibility that knowledge of pollutant concentrations to which subjects have been exposed may bias answers.

Problems of analysis.

There are many problems in analyzing data on pollution and health effects. The studies of Schimmel and his colleagues, Lave and Seskin[4] and the E.P.A.'s CHESS[5] studies and their critical reanalysis make these clearly apparent. Most of these authors have discussed these problems. I do not intend to do so in any detail here. It may, however, be worth saying again that no amount of statistical sophistication (or juggling) can compensate for poor, inaccurate or absent data. It may be very difficult, indeed impossible, to draw conclusions about effects of individual variables when these are highly correlated. Multivariate analysis used to date in pollution studies have always assumed linearity of variates, an assumption which is at least questionable.

The most critical decision from an economic standpoint is, of course, what concentrations should be selected as standards of air quality. On the basis of past epidemiological studies, average 24 hour concentrations of: 250 $\mu g/m^3$ of smoke (as measured by the British smoke filter) and 500 $\mu g/m^3$ of SO_2 are probably reasonable. Average annual concentrations are harder to specify, if indeed such standards are useful. One approach might be to select an annual concentration which experience shows would never permit a 24 hour concentration of more than 250 $\mu g/m^3$ for smoke or 500 $\mu g/m^3$ for SO_2. For London this would result in average annual concentrations of about 50 $\mu g/m^3$ for smoke and 130 $\mu g/m^3$ for SO_2. The main trouble with this approach, however, is that 24 hour distributions of pollutants through the year differ in different places. What might be appropriate for London might not be so for New York City.

A serious problem arises when one tries to convert British smoke concentrations to equivalent total suspended matter (T.S.P.) as measured with a hi-volume sampler, the usual method in the U.S. At relatively high concentration (750 $\mu g/m^3$) the two methods are roughly equivalent; but at low concentrations T.S.P. measurements are roughly three times those measured with a smoke filter. Thus an annual average concentration of 50 $\mu g/m^3$ smoke would translate to about 150 $\mu g/m^3$ T.S.P., a concentration that is about double the current U.S. standard. Most authorities would deprecate relaxing the particulate standard to such a degree without better evidence on the relationship between the two methods. Clearly additional research on the two methods taking particle size into consideration is needed.

These are some of the problems which epidemiologists who are concerned with pollution have to face. There are, of course, many others, such as the cooperation of persons being studied, access to records, completeness of follow-up and so on. But these are general problems for all epidemiological work and the title of the session does say Special. In conclusion, I hope we have not succeeded in convincing you that the problems are so great that epidemiological studies should not be attempted. Epidemiology is the only way of obtaining answers to some of the questions being asked.

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ROLE OF ANIMAL EXPERIMENTS IN RELATION TO HUMAN HEALTH EFFECTS

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INTRODUCTION

When new chemicals are proposed for introduction on the market as food additives, drugs, pesticides, etc., an important portion of the research work done is devoted to evaluation of their possible toxic effects, not only for humans, but also for the biosphere.

Thus, the role of animal experiments is crucial in preventing catastrophic effects to occur. The second role of animal experiments is to investigate and confirm, expand, or deny claims of possible effects on humans of chemicals already on the market and for which epidemiological evidence is introduced about their possible deleterious effects. The most important role of animal experiments is concerned with accurate prediction of safe levels of exposure for the human population.

Animal experiments are also conducted to probe the mechanisms by which toxic reactions are produced, and how the chemicals are biotransformed and eliminated. This established a sound basis for recommendation of safe levels of exposure.

II. REQUIREMENTS OF AN ANIMAL MODEL

In order for animal models to fulfill their role, they must be solidly based on fundamental sciences and provide:

1. Sound anatomical, physiological, or biochemical basis of the measured toxic effects.
2. The response observed must be characteristic, easily recognized, and amenable to quantitation.
3. Concentration-response relationship must be demonstrable.
4. The qualitative correlation between the effect observed in animals and the effect observed in humans must be very high for a series of related chemicals.

5. From the concentration-response relationship, prediction of effects to occur in humans should be made over a wide range of exposure concentrations as well as delineate safe levels of exposure.

Obviously, the above are not always fully obtained with each animal model currently used. Nevertheless, animal experimentation will increase in the coming years and will be refined so that better predictions can be made.

III. SOME ANIMAL MODELS

This Symposium is concerned with health effects from fuel cycle pollutants, both waterborne and airborne. By using some examples of animal models, I hope to demonstrate how they can be used and what their role is, in relation to human health effects.

A. Airborne Irritants

Of industrial and urban atmospheric contaminants, approximately 50% of them have irritating properties. First, they impinge on the surface of the cornea and nasal mucosa and stimulate the nerve endings. This stimulation evokes a burning sensation. Entering the lower airways, these chemicals can also induce bronchoconstriction and inflammatory reactions. Repeated exposure to these chemicals induces chronic bronchitis. An animal model (1,2) has been developed to first categorize airborne chemicals as irritating or not, evaluate and compare their potency, and to predict safe levels of exposure.

a. Basis of the Model

Recognition that an airborne chemical is a sensory irritant is accomplished by monitoring the breathing pattern of an animal, typically a mouse, as shown in Figure 1. When inhaling an irritant, a characteristic pause occurs in the breathing pattern due to stimulation of the trigeminal nerve endings located in the nasal mucosa. A reflex respiratory inhibition is initiated. This response is easily recognized and can be monitored continuously, as shown in Figure 2.

Since the level of response, measured as a decrease in respiratory rate, is related to the concentration of the irritant, a concentration-response relationship can be developed, as shown in Figure 3.

b. Comparisons to be Made

Since concentration-response relationships can be obtained, it then becomes very easy to compare the potency of various chemicals. As shown in Figure 4, the potency of these airborne chemicals varies greatly.

c. Predictions for Human Health

Once concentration-response curves are obtained, a convenient point for comparison, provided the curves are fairly parallel, is the half-way point between no response and maximum response, which for this model can be taken as 50% decrease in respiratory rate and has been termed RD_{50}. The RD_{50} values for the chemicals shown in Figure 4 are listed in Table 1. From these values, predictions of effects to be expected in humans and establishment of safe levels of exposures can be attempted. A list of various standards for exposures to airborne chemicals is in Table 2. In Table 3 are the proposed relationships of RD_{50}

to expected human effects and to the standards presented in Table 2. If we want to test how effective the model is, we can compare the predictions with the current Threshold Limit Value as done in Table 4. We find that for 9 - 11 chemicals, the TLV is between 0.1 - 0.01 RD_{50}. The two exceptions are for epichlorohydrin and formaldehyde. The difference for epichlorohydrin is small, but for formaldehyde, the acceptable range for TLV predicted by the model is much lower. This difference has been discussed and is due primarily to the fact that the animal model does not consider tolerance development to this chemical, while in workers continuously exposed to formaldehyde, this phenomenon occurs (3).

Another test of the predictions made by the model is to evaluate how accurate the predictions are for the range of exposure concentrations. This is presented for 2 of the 11 chemicals, acrolein and chlorine in Tables 5 and 6 (see Reference 1 for others). It can be seen that, in general, the predictions made within each concentration range are reasonable. Unfortunately, for the lowest concentration, with the exception of sulfur dioxide, there were no data in the literature to permit us to present firm conclusions. However, as long-term chronic studies on these chemicals are being conducted, these results should complete the picture.

d. Difference in Sensitivity

In predicting levels of effects for humans, we must take into account, the difference in sensitivity existing in the population. This is particularly difficult to take into account in animal experiments since we are using fairly homogenous pupulations. Several species can be tested, or different strains can be tested to obtain an estimate of the variation in sensitivity. This has been done using the model described above and some of the results are presented in Table 7. As can be seen, there is a factor of 10 between the most and the least sensitive strains. Such variations can then be taken into account when making predictions of human reactions from animal models.

B. Pulmonary Hypersensitivity

Many industrial contaminants can elicit bronchial hypersensitivity. Reports of asthma-like attacks by workers exposed to simple chemicals such as toluene diisocyanate, phtalic anhydride, trimellitic anhydride, ethylene oxide, formaldehyde, ethylene diamine, etc., are frequent. Exposures to these chemicals stimulate the formation of reaginic antibodies.

a. Basis of the Model

Several animal species have been used to investigate the sensitizing properties of airborne chemicals. When challenged with an aerosol of antigen, a characteristic breathing pattern results which can be quantitated so that the magnitude of the response can be obtained (4). Using the challenging antigen, sera from sensitized animals can then be tested for antibody reactive toward the hapten groups (i.e., specific chemical) of interest.

b. Application in Humans

Once it has been demonstrated that the antigen prepared can be used to detect antibodies in sera of animals, it becomes very easy to apply the technique for evaluating sera from workers exposed to the chemical, in this case, toluene diisocyanate (5,6). Such tests, as shown in Figures 5 and 6 can identify workers with elevated levels of IgE antibodies and permit the follow of their disappearance, once the workers are removed from exposures.

IV. CONCLUSIONS

Society is now entrusting toxicologists with the primordial responsibility to prevent entry on the market of chemicals which can lead to disastrous effects on a very large scale. Often we read newspaper accounts, as well as scientific reports of adverse effects of chemicals. Have toxicologists failed to protect the public? What we do not read about is the number of promising chemicals which are prevented from entering the market because of what toxicologists found in their investigations using animals. It would be interesting to have some accurate statistics in this area to determine the performance of toxicologists and the animal models used.

It remains, however, that toxicologists will be at the front-line of evaluation of new chemicals and it is only via toxicological testing in animal models that we can prevent adverse effects to occur in man.

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TABLE 1

RD_{50}* and 95% Confidence Limits for Eleven Sensory Irritants

Compounds	RD_{50} ppm	95% Confidence Limit ppm
Acrolein	1.68	1.26 - 2.24
Ammonia	303	159 - 644
Chlorine	9.34	6.64 - 14.1
Chloroacetophenone	0.96	0.766 - 1.26
Chlorobenzylidene malononitrile	0.52	0.429 - 0.677
Chloropicrin	7.98	6.22 - 10.6
Epichlorohydrin	687	633 - 748
Formaldehyde	3.13	2.54 - 3.97
Hydrogen chloride	309	281 - 410
Sulfur dioxide	117	107 - 128
Toluene diisocyanate	0.39	0.345 - 0.446

*RD_{50}: Exposure concentration associated with a 50% decrease in respiratory rate.

TABLE 3

Proposed Relationships of RD_{50} Concentration Values to Industrial and Environmental Standards

Concentration	Designation	Expected Response in Humans	Proposed Relationships to Industrial Standards	Proposed Relationship to Environmental Standards
10 RD_{50}	Lethal	Lethal or extremely severe injury to the respiratory tract	——	——
RD_{50}	Toxic	Intolerable sensory irritation; respiratory tract injury may occur with extended exposure	——	——
0.1 RD_{50}	Effective	Definite but tolerable sensory irritation	Highest acceptable concentration for TLV 0.2 RD_{50}-basis for STEL 0.3 RD_{50}-basis for EEL	——
0.01 RD_{50}	Ineffective	Minimal or no sensory irritation	Lowest concentration necessary for TLV	——
0.001 RD_{50}	Acceptable	"Safe" level of no effect	——	Recommended highest concentration for Air Quality Standard

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TABLE 4

Prediction of Sensory Irritation Responses in Humans for Eleven Sensory Irritants Evaluated in Mice, and Relationship of RD_{50} to Current TLV-TWA Values*

Sensory Irritant	Predictions of Responses in Humans			1977 TLV-TWA ppm	Is Current TLV-TWA between 0.1-0.01 RD_{50}
	Intolerable at RD_{50}(95% C.L.) ppm	Uncomfortable but Tolerated at 0.1 RD_{50} ppm	Minimal, No Effect at 0.01 RD_{50} ppm		
Acrolein	1.68 (1.26-2.24)	0.2	0.02	0.1	yes
Ammonia	303 (159-644)	30.0	3.0	25	yes
Chlorine	9.34 (6.64-14.1)	0.9	0.09	1	yes
Chloroacetophenone	0.96 (0.766-1.26	0.1	0.01	0.05	yes
Chlorobenzylidene malononitrile	0.52 (0.429-0.677)	0.05	0.005	0.05	yes
Chloropicrin	7.98 (6.22-10.6)	0.8	0.08	0.1	yes
Epichlorohydrin	687 (633-748)	70.0	7.0	5	no
Formaldehyde	3.13 (2.54-3.97)	0.3	0.03	2(C)*	no
Hydrogen chloride	309.0 (281-410)	30.0	3.0	5(C)*	yes
Sulfur dioxide	117 (107-128)	12.0	1.2	5	yes
Toluene diisocyanate	0.39 (0.345-0.446)	0.04	0.004	0.02(C)*	yes

* (C) indicates Ceiling Value

TABLE 5

Acrolein (3)

Predicted Effect According to Proposed Model	Concentration Factor in Terms of RD_{50}	Corresponding Concentration ppm	Reported Exposures to Acrolein (Results are for humans unless otherwise noted)	
Lethal or extremely severe injury to the respiratory tract.	10	20	21.8 ppm	Intolerable.
			10.5 ppm x 6 hr	1/2 rabbits died.
			8 ppm x 4 hr	1/6 rats died.
			5.5 ppm	Intense irritation.
Intolerable sensory irritation; respiratory tract injury may occur with extended exposure.	1	2	1.2 ppm x 5 min	Extremely irritating - only just tolerable.
			1.2 ppm x 5 min	87% of test panel reported irritation.
			1 ppm x 5 min	82% of test panel reported irritation.
			1.8 ppm x 30 sec	Odor detectable.
			1.8 ppm x 4 min	Profuse lacrimation, practically intolerable.
Definite but tolerable sensory irritation. Highest acceptable concentration for TLV.	10^{-1}	0.2	0.5 ppm x 5 min	19-35% of test panel reported irritation.
			0.5 ppm x 12 min	91% of test panel reported irritation.
			0.3 ppm x 60 min	Considerable acute irritation after 10-20 min; decreased respiration.
			0.15 ppm	Nasal irritation.
Minimal or no sensory irritation. Lowest concentration necessary for TLV.	10^{-2}	0.02	0.09 ppm	Eye irritation.
			0.06 ppm	Eye irritation 0.471 on scale of 2.0.
Safe; level of no effect. Recommended highest concentration for Air Quality Standard.	10^{-3}	0.002		No data available.

TABLE 6

Chlorine (1)

Predicted Effect According to Proposed Model	Concentration Factor in Terms of RD_{50}	Corresponding Concentration ppm	Reported Exposures to Chlorine (Results are for humans unless otherwise noted)	
Lethal or extremely severe injury to the respiratory tract.	10	90	1050 ppm	Lethal concentration.
			1000 ppm	Rapidly fatal.
			105 ppm	Tolerable for only a few seconds.
			40-60 ppm	Dangerous for even short exposure.
Intolerable sensory irritation; respiratory tract injury may occur with extended exposure	1	9	20(8.7-41) ppm	Painful eye irritation.
			16.8 ppm	Irritation of throat in 3 minutes.
			14-21 ppm	Dangerous.
			9(2.6-41) ppm	Intolerable respiratory irritation.
			5 ppm	Chronic exposure caused premature aging; bronchial disease; mucous membrane inflammation; corrosion of teeth.
			3-6 ppm	Stinging or burning of eyes, nose, and throat; headache.
			3.0(1.9-4.2)ppm	Painful respiratory irritation.
Definite but tolerable sensory irritation. Highest acceptable concentration for TLV.	10^{-1}	0.9	1-2 ppm	Men can work.
			1.3(0.02-2.9)ppm	No eye or respiratory irritation.
Minimal or no sensory irritation. Lowest concentration necessary for TLV.	10^{-2}	0.09	0.05 ppm	Odor detectable.
Safe; level of no effect. Recommended highest concentration for Air Quality Standard.	10^{-3}	0.009		No data available.

TABLE 7

Results of Sensory Irritation Obtained in Various Strains of Mice with Exposure to Sulfur Dioxide

Strains	Sex	H-2 Haplotype	RD_{50}* (ppm)	95% C.L.
A/HEJ	F	a	41	39-44
A/HEJ	M	a	69	46-104
BALB/C+	F	d	75	72-80
BALB/C+	F	d	78	53-114
BALB/C	M	d	106	67-167
C57/BL6	F	b	80	59-110
SJL/J	F	s	104	72-150
SJL/J	M	s	320	162-630
C3H/HEJ	F	k	125	110-142
C57L/J	M	b	200	113-352
DBA2/J	M	d	321	178-580
DBA2/J	F	d	445	333-595
A/J	F	a	> 450	---
Swiss ø	M	-	117	99-138
Swiss	M	-	117	67-204
Swiss	F	-	133	94-187

*Value obtained from concentration-response relationship which represents the concentration necessary to obtain 50% decrease in respiratory rate. Least squares linear regression analysis with F test for regression significant at 0.05 level for all groups.

øResults from previous report.

+Data obtained from two different shipments--six months apart.

From Alarie, Y., Oka, S. and Cypess, R., unpublished, under NIEHS grant #ES-00872.

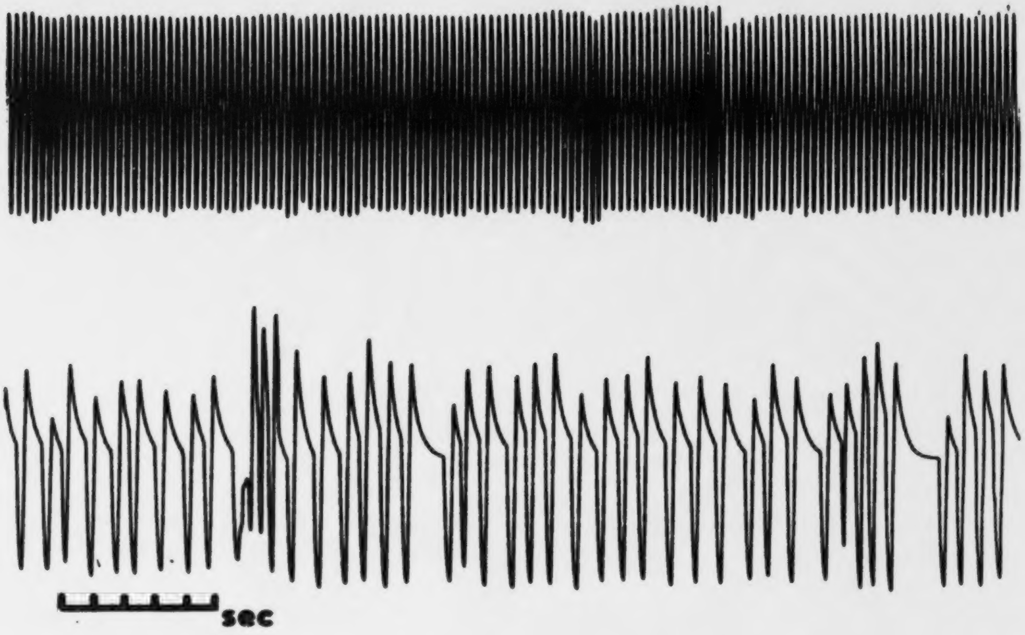

Figure 1 - Typical oscillograph display showing the respiratory cycle of a mouse during normal control conditions (upper tracing) and exposure to a sensory irritant (lower tracing).

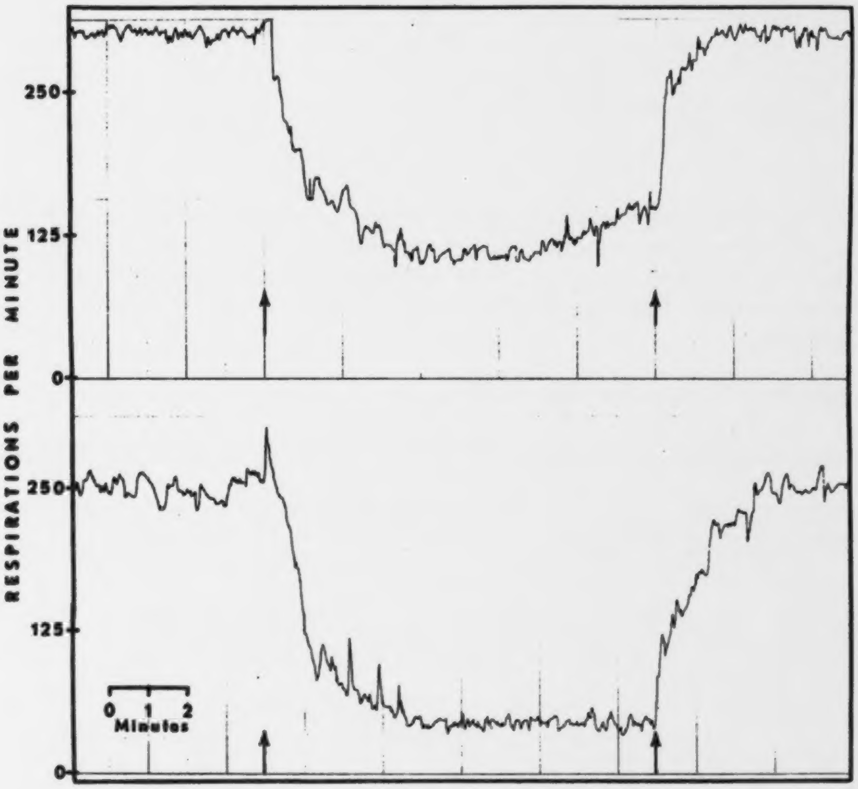

Figure 2 - Average respiratory rate of 4 male Swiss-Webster mice prior, during, and following exposure to formaldehyde (top) and acrolein (bottom).

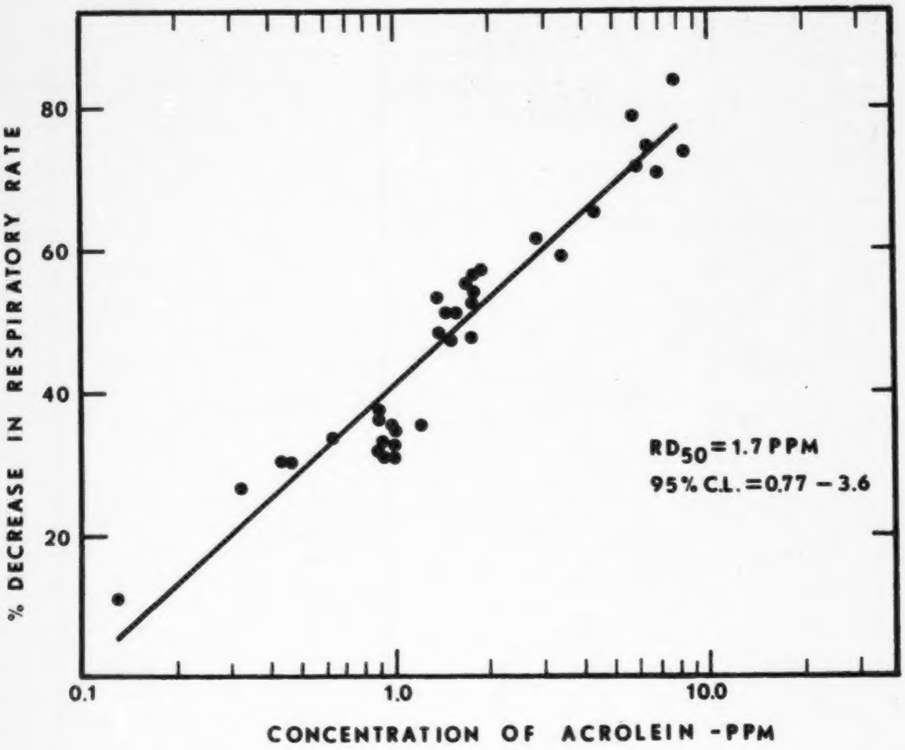

Figure 3 - Concentration-response relationship obtained with exposures to acrolein in male Swiss-Webster mice.

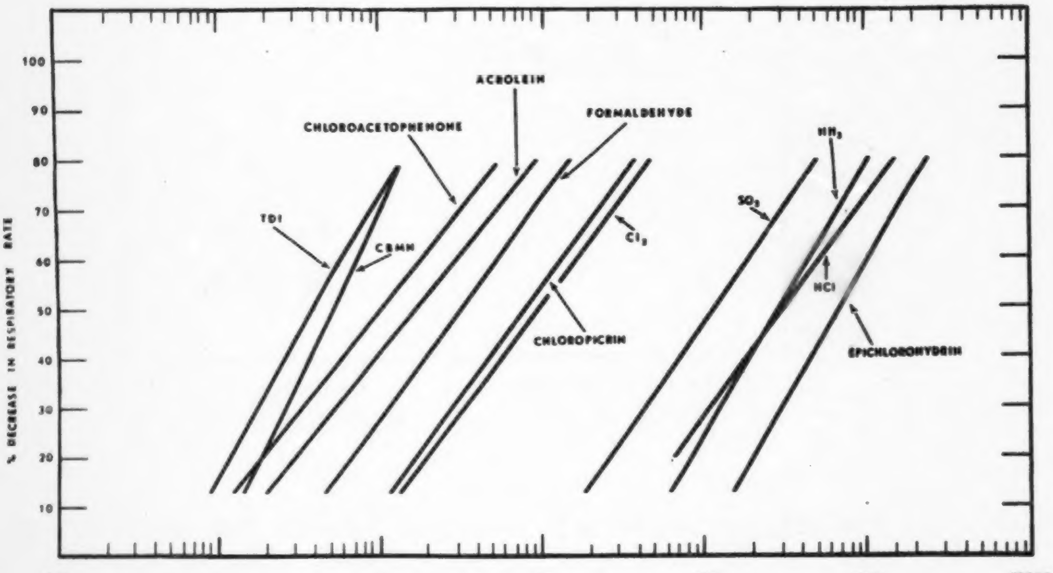

Figure 4 - Concentration-response relationships obtained for eleven sensory irritants in male Swiss-Webster mice.

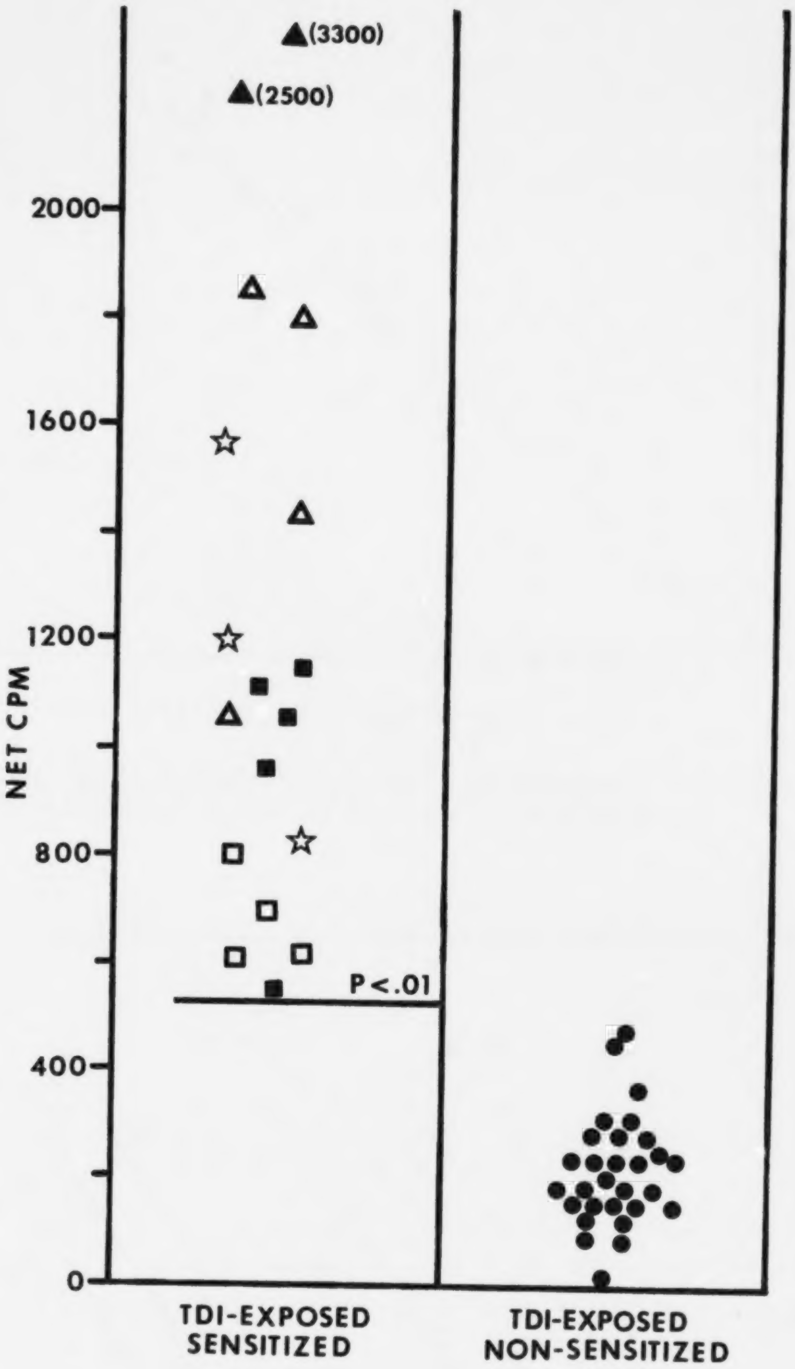

Figure 5

RAST assay for tolyl-reactive IgE antibodies in sera from workers exposed to TDI. Net cpm = cpm (T-HSA discs)-cpm (HSA discs). Clinical reactions upon TDI exposure: ▲ , immediate response confirmed by bronchial challenge; △ , immediate bronchial response; □ , delayed bronchial response; ■ , cutaneous response; ☆ , delayed bronchial and cutaneous responses; ● , no response. Significant titers: $>$ 456 net cpm ($P < 0.01$), $>$535 net cpm ($P < 0.001$).

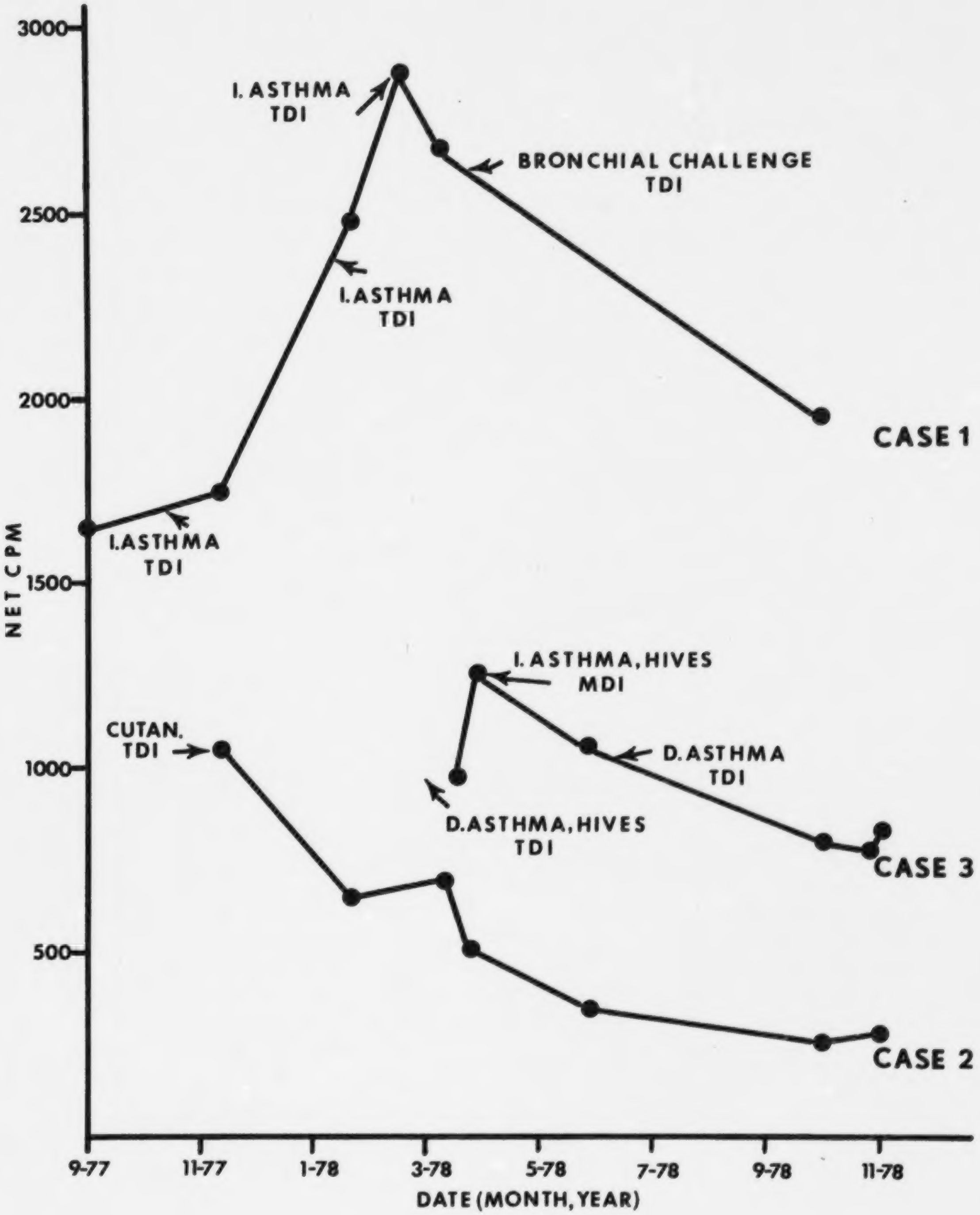

Figure 6

RAST assay for tolyl-reactive IgE antibodies and clinical hypersensitivity episodes in 3 workers with TDI sensitivity. Net cpm = cpm (T-HSA discs) - cpm (HSA discs). I. Asthma: immediate bronchial reaction occurring within 15 minutes of exposure. D. Asthma: delayed-onset bronchial reaction occurring more than 120 minutes following exposure.

DISCUSSION

SESSION II

A question to Dr. Higgins: Regarding one of the areas of the pure type of pollutants, that originally needs epidemiological assessment. As you know, a cornerstone of the national energy program is the dieselization of mines. Because of substantial increases in coal mined daily, each mine has opted for dieselization. However, the Unions have gone on record against dieselization. Just last week the West Virginia legislature passed a moratorium on all diesels in West Virginia mines for two years or until such time as the health impact of use of diesels in mines has been resolved. Since the health effects of diesels may be a big problem I would like to solicit from Dr. Higgins or other members of the panel any comments or suggestions they may have in regards to this urgent problem.

Dr. Higgins: The use of diesels underground and elsewhere has become a political rather than a scientific issue. This is a pity because there are important scientific questions to be answered about the hazards of diesel emissions. There are two main worries: do the emissions cause cancer of the lung? and do they result in chronic obstructive lung disease? The evidence, such as it is, suggests that they are not serious hazards for either of these conditions. No study of workers exposed to diesel emissions has shown an excess cancer risk. Some studies of chronic respiratory symptoms and disability have suggested some effect; but others have not. In Dr. Rockette's study of the mortality of miners, mortality from lung cancer was highest in Charleston, West Virginia, an area where diesels are not used.

Dr. Hamilton: I don't wish to change the topic immediately on diesels, because I think there's an area of uncertainty here. But I want to ask Dr. Lippmann a question because I have the general impression from his talk that he felt very comfortable with the current methods of assessing the conventional air pollutants, the health effects of which we seem to have a great debate about. One of the problems is exemplified, for example, in the recent Congressional review, it isn't so recent now, in a review of the CHESS study they were

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very critical of the methods of measurement. Do you feel that existing methods of measurement of the conventional air pollutants, particularly of the species of the oxidation products of sulfates, are such that we can place reliance on them and later judging about the possible health effects assessment?

Dr. Lippmann: I think there are really several questions. One, I indicated that the only sulfur oxide we know how to measure reasonably well was sulfur dioxide. We don't know how to make routine measurements of any of the sulfur oxide particulates directly. In fact, we rarely measure them effectively on collected filter samples. If you want to measure sulfuric acid, you have to use teflon filters and do chemical separations prior to the analyses. Clearly, we don't have adequate methods for many of the pollutants of interest. Now, sulfuric acid is not currently a regulated material; i.e.,there is no ambient air standard for it. That is perhaps one reason why we have not seen any effective instrument development, although EPA has supported several instrument development projects which were unsuccessful. While I agree with you, in general, that we don't have accurate measurements, the fact that we don't end up with good measurement and data bases is seldom the problem of the technology. It's having the foresight to know what to measure, and to have the resources, financial as well as technical, to make enough good measurements.

Question: My first question, for Dr. Lippmann, is really a follow-on to the one he just answered. Would you comment on the actual field experience in the use of measuring techniques, going from the technological capability to what actually has occurred? My second question is to Dr. Higgins. Would you comment on the influence of the actual field experience in the use of measuring techniques? The reason for this question is clearly that if you are investigating diseases that have latent periods of 20 or 30 years, and you do a ten year study, you're clearly going to get nothing, but this fact does not prove anything. You may have spent a million dollars of the taxpayers' money with no result. The final question that I have is for Dr. Alarie: Would he comment on the Ames test as a method of detecting carcinogens, that being a kind of animal model to which human experience is now being compared.

Dr. Lippmann: A real problem in any study involving human investigators and technicians is misuse of instruments. Instruments may have good designs and demonstrated feasibility, but that doesn't mean that you can depend on the technicians who are likely to be employed in moderately difficult situations to use them properly. The only reliable data are from studies where there are elaborate quality control programs behind them to make sure that the calibrations are performed frequently and reliably and, if necessary, where corrections or adjustments can be made in the instruments. There is one other point. I think you can be misled by worrying about the precision of the data. As Dr. Higgins pointed out, the sampler is seldom located in the most appropriate place, and worrying whether you have a 10, 20 or 40% error in the measurement may be excessive. The expense of improving the precision of measurement at that particular site may not be worth it when the site is not representative of the area in which it is located.

Dr. Higgins: Two years ago when we were studying air pollution in Florida, we learned that the West Gaeke method for measuring SO2 could be completely invalidated if the temperature in the sampling box rose to high levels. Here was an example of a problem with a test which has been used for many years as a standard method which had been completely ignored--until a technician brought it to light.

With regard to duration of follow-up, clearly a long period may be necessary if you are to identify cases of cancer with a long latency. I have never been completely convinced, however, that you will get nothing from shorter periods of follow-up. I suspect that some more sensitive subjects should show up after shorter periods. Clearly, if you are studying a population with about 20 years since first exposure to a suspect carcinogen if the average latency period is 25 years, 20 years will not be long enough to identify all the cancer cases which may develop. The next 10 or 20 years may be crucial. 20 years may, however, identify some excess cases. It is clearly very important to live long enough to do the further follow-up or at least to will the data to someone who will do it posthumously for you.

Dr. Alarie: Well, I have no problem with the Ames Test. I think it's entering into a sort of second generation now, where potency response or dose response curves are being obtained, which I have shown in the model we have. You can't go anywhere in terms of prediction until these are done and you have it for several carcinogens on which you have some human data. We have several carcinogens, known carcinogens, in man from extremely potent to moderately potent and some probable. One would think that Ames is intelligent enough to recognize that fact. As a matter of fact, I think he is doing just that right now. So I predict that within two to three years it certainly will be a quite useful test. Right now for yes or no answers that is ridiculous. Secondly, there are a lot of idiots running around dumping a lot of things on the Ames test knowing absolutely nothing about what they're doing. And they're publishing letters here and there and everywhere saying it's no good. If you dump a highly reactive chemical in these nutrient systems and then they come out negative; well, what you have is a stupid test to begin with.

Dan Swartzman, University of Illinois School of Public Health: I have a question for the panel or the attendees or the moderator. I am troubled by the use of indicators, although I understand it is a rather useful scientific shorthand. I'm reminded of the situation with guidelines on wage and price control where you set a 7% ceiling, which all of a sudden becomes the target that everybody tries to meet. In community air pollution control we've found that setting an indicator for ozone as the chemical indicator for the control of chemical oxidants concentrated our efforts on solving the ozone problem, not on solving the photochemical oxidant problem. To the extent that recently USEPA has decided that, in fact, the photochemical oxidant problem is an ozone problem. Similarly with the use of sulfur dioxide as an indicator of whatever. The total suspended particulates-sulfur dioxide complex problem, using the sulfur dioxide as an indicator of that, has led to a lot of political problems, particularly in my home state of Illinois where we're trying to do the best to protect the public from health problems but we are way below the sulfur dioxide levels. We get a lot of people saying, "Well, you can have a great deal more sulfur dioxide without doing harm to the general public. Look how far below the levels we are." I'm troubled by the process, the political process by which the indicator loses its effect as a symbol and instead gains some respectability as the end in itself. I was wondering if anybody has suggestions, aside from just constantly reminding ourselves and the politicians and bureaucrats that these were established for use as indicators.

Dr. Lippmann: I could not agree with you more--in fact, I spent my time pointing out that the most commonly used indicator for air pollution potential has no relevance. Sulfur dioxide is the best example possible. Because we did not know what component of the complex to measure, we measured the one that we could most easily measure. On the other hand, what should one do when you have such a complex mixture and you can't possibly measure all the potential toxicants? The only useful thing to do is to keep the limitations of the measurements in mind and to pick an appropriate indicator. For example, ozone appears to be an appropriate indicator for the photochemical smog mixture, albeit with some limitations. SO2 is a poor indicator for the SOx-particulate complex because SO2 is essentially non-toxic at realistic ambient levels. I think having a group of indicators specific to the process may be the most positive thing we can do in the control sphere at this time.

Dr. Hamilton: I'd just like to ask Dr. Lippmann to clarify his last remark. My understanding of SO2 is that it is a precursor of sulfate.

Dr. Lippmann: Sulfates form continuously and gradually over long distances and I think a rational national policy would be to limit sulfur dioxide emissions over the whole Northeast in a uniform way. To have certain cities use 0.3% sulfur fuel and other cities use 3.0% sulfur fuel is completely irrational.

Dr. Hamilton: I couldn't agree with you more except that you're limiting it to the Northeast. It seems to me that with prevailing winds we should certainly put the major emphasis further west.

Dr. Lippmann: I didn't mean the Northeast in a limited sense--I meant the whole Northeast quadrant of the United States from Minnesota eastward and perhaps we should include the southeastern part of the country as well. We have a different problem out west than we do in the east.

Swartzman: I'd like to just clarify the problem I see with the sulfur dioxide particularly in our state, (Illinois). We're probably on the western edge of prevailing winds that created the sulfate problem in the rest of the country. We have a number of power plants around the state that are located in areas in which the ambient air quality is far below the sulfur dioxide national ambient air quality standard that has been set. There is a great deal of movement in our state, by the utilities, by the coal industry, and some other right thinking people, who feel we ought not to have that sort of "overkill" because we're so far below the standard that these plants ought to be allowed to emit more sulfur dioxide than they are currently allowed to emit. The problem that faces public health advocates is that now that we have got this wonderful indicator that tells us what the problems are, we're stuck with it as the problem itself. We're so far below the sulfur dioxide level in some of these cases that it is absolutely impossible to argue for lower emission levels on a particular plant. Particularly when the people in the legislature are able to say, "Well, it's not our problem--it's New York's problem--let them do something about it." That is the problem that we run into, and I hate to see us develop the same problem with coal conversion technology. Illinois is a prime state for placing coal conversion plants. I think that again if we start concentrating on those indicators without having some definite method of making sure that throughout the whole process we maintain our focus on the real problem, we will create more problems than we solve.

Dr. Lippmann: The model is really what EPA did with auto emissions. You can't set ambient standards for everything, and sometimes we control the things that can become air pollutants, like converters at the source. I think the intelligent thing is to direct the control technology at the source and not through the ambient air quality standards. I think you're making a great mistake, which I suspect they will, about using total sulfate as the next air quality standard to drive controls. Because ammonium sulphate is innocuous and that's where most of the measured sulfate will be. Sulfuric acid is something else again, because of the very variable percentage of it. Sulfates might be important , but we just don't have enough data base to really know. But total SO4 measured on a filter, I think would be just as misleading, and just as misguided, as an engine to drive controls of SO2 gas.

W. C. Hamilton, Continental Oil Company: I wonder if Dr. Higgins would expand on his asthma remarks.I talked to an asthma specialist last week who pointed out that not only do foods and pollens exacerbate asthma attacks but there is a strong psychogenic component and temperature change that have an effect. He said that while he did not know anything about epidemiology, he did feel it would be very difficult to ascribe any asthma attacks to air pollution per se. It might occur or might not occur, but one could never tell.

Dr. Higgins: I agree, it is difficult to acsribe asthmatic attacks to air pollution, but I don't believe it is impossible. In fact, asthmatic subjects may respond rather specifically to certain substances. The trouble is that most asthmatics respond to many different stimuli. The problem is to identify asthmatics who respond specifically to pollutants and then try to assess the consistency of their response.

Another problem with asthmatics and air pollution is that there may be a response to some pollen which has not been measured but which may vary in parallel with pollution.

A further problem is that asthmatic attacks may be triggered by short peaks of pollution which may have very little effect of the 24 hour pollutant concentration. It might appear in such circumstances that a very low 24 hour concentration is causing attacks and that consequently no concentration of the pollutant is acceptable. On the whole, asthmatic attacks are in my view a dangerous way of reaching acceptable pollutant concentrations.

Question: Is there any way you can measure the sulfuric acid in the emissions?

Dr. Lippmann: From a point source, the answer is yes. There are quite acceptable methods for stacks or other point sources where you have relatively high concentrations. I am less sanguine about the possibility of measuring a few micrograms to 20 micrograms per cubic meter in ambient air; there you may have some problems. If you are influenced by a local point source it could be measured by available technology with pretty good precision by someone in your local health agency who could help.

Fred Lippert, Brookhaven National Laboratory: I want to continue this debate I thought we had started on the sulfur dioxide standards. I am a little confused as to whether there is a disagreement among members of the panel or not. Dr. Higgins, I believe, said he could live with 100 micrograms annual average of SO2 . I presume that included the sulfate that goes with it. And Dr. Lippmann, if I understood you correctly, you want to put the whole country on 0.2% sulfur or do you want to hold that to 2%? I am not sure which you meant. You're concerned about sulfuric acid, and as a result you are willing, regardless of the cost, to eliminate all the sulfur in the atmosphere. Can you straighten me out?

Dr. Griffin: To whom are you addressing the question?

Fred Lippert: I'd like to hear if they both agree on this or if they disagree.

Dr. Higgins: On the basis of the epidemiological evidence and even more from animal experiments it is difficult to support an ambient average annual standard fo SO2 of less than 100 microg/m3. Alone, SO2 appears remarkably harmless. One must remember, however, that such an average annual concentration is compatible with a one to three hourly concentration of perhaps 2 mg/m3. This puts a somewhat different complexion on the issue. Furthermore, SO2 does not usually occur alone, and may be converted into other chemicals--sulfates, for example, which have sometimes been thought to be more irritant, though there is now doubt about this.

Dr. Schimmel has been studying daily deaths in relation to daily SO2 and particles in New York City. He believes SO2 to be not only harmless but even beneficial. I find it difficult to go along with him on this. But it is clearly important to try and resolve the issue.

Dr. Alarie: My grandmother knew that. Dr. Higgins, I am against this primitive method of burning sulfur candles at the wake as a sort of healthy activity of helping people to the next world.

Dr. Lippmann: I was not advocating any particular level--I was simply trying to get the thinking going in a different direction. While SO2 by itself is not really likely to be important, it does act as a messenger. In the past we attempted to control the messenger rather than getting at the real problem. It is time to get past that type of thinking. The visibility problem is largely a sulfur oxide particulate problem for the northeast quadrant of the United States and for the southeast to some extent also. In terms of acid rain, we also have a problem which has to be addressed by control of sulfur emissions. What I'm saying is that it's not fair to put the burden of emissions controls on areas of the country selectively for what will turn out, in retrospect, to be capricious reasons. I don't know what percentage of sulfur emissions control is appropriate for this whole quadrant of the country where more than 50% of the population lives. But, it should be distributed more equitably than it is now. It is likely that 2% is too high, and 0.3% is too low for all sources. We will have to come to some compromise figure, which, as Senator Bayh suggested, may not necessarily be a zero risk level. But it, at least, should be more equitable than the multiplicity of limits now used in the various local regions.

Dr. Griffin: Let me ask one clarifying question. Are you suggesting that the control of air pollution should not be based on attempting to deal with air quality standards of some sort locally? Or rather, that our control should be on an industry-wide basis, so that there should be a reduction in emissions of all sorts? Is that the basis for your suggestion?

Dr. Lippmann: I won't say that across the board; I do say so for sulfur dioxide which is transformed to sulfates during long range transport. We, for instance, in our own air pollution studies, find that a location at High Point, New Jersey, a very lightly populated site at the confluence of New York, New Jersey and Pennsylvania in the summer-time, has sulfate levels which are 85% as high as they are in New York City. Thus, the contribution of the metropolitan region to the sulfates measured in New York City, is only about 15 to 25%. In that kind of situation the concept of local air sheds is completely inappropriate. For other pollutants, it may still be appropriate.

Julian Andelman: I have a comment and a question. As far as standards are concerned, in the water field they are different and they can be useful, particularly for those parameters that may not be getting to levels that could be harmful regularly, but rather when they're normally low and then become indicators of the central problem. I feel more comfortable with using them not necessarily for control levels, but as indicators of the problem; for example, carbon chloroform extract as an indication of high levels of organics in water.

In 1970 the World Health Organization adopted the guideline for six polycylic aromatic hydrocarbons in drinking water, such that when the total concentration exceeded 20 ug/l, this would be reason for concern and further investigation. And today in the water field there is a lot of concern about chlorinated organics, some of which one could readily measure but a full scale set of analysis would be rather complex; thus people are trying to find indicator parameters, like total organic chlorine, which one could perhaps more easily monitor. Hopefully, this would ultimately result in a set of standards, but for now the measurement simply would be used as a guide for levels of concern.

I have a question I'd like to address to Dr. Alarie to perhaps expand on a major point he made. How valid might this target be for those chemicals that are sensory irritants, but might also have other toxicological properties, like latent effects on the central nervous system, or might be mutagens or carcinogens? In other words, might this be expanded to those sensory irritants that have more serious or more variable human effects?

Dr. Alarie: I'm talking about a concept of getting a concentration response curve and obviously the concept applies to any model system you want to use, not only the model we have used. If you have a chemical, let's say carbon monoxide, and this animal model is used, the animals will die and no irritation response will be seen. Any model has limitations. However, basically any chemical can be placed into a classification; I've shown one model for sensory irritants and developed one for pulmonary hypersensitivity and one can be developed for any endpoint that you are interested in. I haven't talked about limitations of models. We're working on this now with 11 very common solvents that are used in industry, and where the RD50s are now in the thousands of parts per million. At one point, I fully expect that the model will no longer be useful since another toxic effect will predominate.

Dr. Lippmann: I think though that there is one point in your comments which really does stand out and has to be addressed. If the material is a sensory irritant, and only a sensory irritant, the model is great. When the sensory irritant is also something else, such as a carcinogen, clearly it is not great. Epichlorohydrin, in particular, has been shown to be an animal carcinogen by various routes of administration, including inhalation, and I believe that the results from epidemiological studies are pointing in this direction also.

Dr. Alarie: That's fine with us. We have no objection that chemicals would have other effects--obviously we expect that they will have other effects. What we are interested in is predicting a level, a concentration, at which epichlorohydrin will not induce cancer. At the Society of Toxicology last week, there was a dose response relationship presented for the carcinogenicity of epichlorohydrin. This came from a 90 day study and there is now a three year study going on. Therefore, I'm looking forward to see at what level we are going to detect cancer occurrence. This would be true also for bischloromethylether. Bischloromethylether is an extremely potent sensory irritant, also the most potent airborne carcinogen known in man. But it also has a dose response relationship. It also has a concentration at which you see no cancer occurring, as has been shown in animal studies.

Dr. H. Spencer: One comment about laboratory animals. Dr. Alarie, could you tell us what the impact on the public would be if we used rabbits to test the edibility of mushrooms?

Dr. Alarie: I'm sorry, what do you want to test?

Dr. Spencer: I would like you to tell me what the impact would be on the public if we used rabbits to test the edibility of mushrooms?

Dr. Alarie: We use rabbits? Rabbits to test what?

Dr. Spencer: To test the edibility of mushrooms. What would happen to the public?

Dr. Alarie: Why do we use rabbits to test the toxicity of mushrooms?

Dr. Spencer: The point I was trying to make is that the panel apparently assumed that the concept of biochemical unity is infallable, which is obviously not correct. Rabbits can eat toxic mushrooms and survive. The point being that some chemicals you may test by animal model, and that animal could well have no response whatsoever.

Dr. Alarie: You see the way you address it, of course, is you don't pick up one single species of animal. We have a wide variety of them. There is no problem whatsoever and I certainly can find one man on the face of this earth who will look exactly like your rabbit. But the problem is he will be the exception. So what I want is an animal which will represent the human population. I go searching for it by trials and errors. It is not something I can guess.

Joe Banon, Chicago Lung Association: I was riding home in my car from work about a month ago, and I heard a news report on an air pollution study, to be done by the Yale Lung Research group, where two towns in Connecticut, I believe, are used to study the prevalence of respiratory diseases. Dr. Higgins, if you are familiar with the study, can you comment on it?

Dr. Higgins: Yes, I am familiar with the studies you mention. I think one of the main difficulties is that the differences in pollutant levels between the two towns was very small. One would not expect to be able to show differences in bronchitis, lung function, etc., due to pollution because even the more polluted town was not polluted enough. We had a similar problem in our studies in Florida.

SESSION III: HEALTH ASPECTS OF FOSSIL-FUEL ELECTRIC POWER PLANTS

Tuesday morning, March 20, 1979

Moderator: James J. Stukel, Ph.D.
Professor of Civil Engineering
University of Illinois

AIR POLLUTION

By
Benjamin G. Ferris, Jr., M.D.

HUMAN EXPOSURES TO WATERBORNE POLLUTANTS FROM COAL-FIRED STEAM ELECTRIC POWERPLANTS

By
Julian B. Andelman, Ph.D.

OCCUPATIONAL HEALTH ASPECTS OF FOSSIL-FUEL ELECTRIC POWER PLANTS

By
William N. Rom, M.D., M.P.H.

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AIR POLLUTION

By

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HEALTH ASPECTS of FOSSIL-FUEL ELECTRIC POWER PLANTS - AIR POLLUTION

Benjamin G. Ferris, Jr.

Fossil fuels used for electric power include coal, oil and gas. Coal and oil make up the major sources of energy. As a result of their combustion, particulates, sulfur oxides and oxides of nitrogen are produced. Coal can produce large amounts of fly ash and soot which in turn can be largely controlled by various methods. Oil usually does not produce so much. Sulfur dioxide production will vary with the sulfur content of the fuel. This component is more difficult to control than the fly ash. Nitrogen oxides are also generated and when diesel engines are used they can make considerable impact on ambient levels. They are even more difficult to remove from the emissions than SO_2. Control has been directed to limiting the generation of NO_2.

These components consist of the major air pollutants from fossil fuel. This presentation will discuss their apparent impact on the health of human beings with special emphasis on levels at somewhat above the present air quality standards.

OXIDES OF NITROGEN

NO and NO_2 are produced due to the heat and pressure if present, as in diesel engines. NO appears to have little effect. It can be converted in photochemical smog to NO_2 which is reactive. Thus this discussion will be on the effect of NO_2.

Chamber Studies - Short-term

A number of chamber studies for short-term exposures have been done.

These are summarized in table 1. Most of these have involved healthy subjects (1, 5, 6, 13). A few have used asthmatics (7) or "reactive" subjects (3). Some have used mixtures of pollutants (4, 14). Most of these studies have produced negative results if levels of NO_2 have been below 2.82 mg/m^3 (1.5 ppm). One study (7) did indicate that 3 out of 20 asthmatics showed a measurable increase in airway resistance after a one hour exposure to 0.2 mg/m^3 NO_2, and 13 out of 20 had an increased sensitivity to carbacol. The subjects apparently did not detect any change in their airway resistance. This study has been criticized because of the varying levels of the baseline airway resistance in the subjects. Furthermore, the relevance of these sorts of observation to the real world conditions have also been questioned. Certainly further studies of this sort and in this dose range are warranted.

Kerr and coworkers (6) have exposed 13 asthmatic, 7 chronic bronchitics and 10 normal adults to 0.940 mg/m^3 (0.5 ppm) NO_2 for 2 hours in a chamber. A 15-minute period of moderate exercise was performed during the first hour of exposure. Each person had a 2-hour control period in the chamber on the day before the exposure. They did a variety of tests of pulmonary function including airway resistance. The study was not made double blind since NO_2 could be detected by odor at this level. During the exposure to 0.940 mg/m^3, 7 of the 13 asthmatics had varying symptoms - two burning of their eyes, one headaches, and from some, chest tightness on exercises; one normal and one chronic bronchitic had a running nose. All of these symptoms were felt to be slight. No delayed reactions were reported. No significant changes were noted in the various measured parameters (spirometry, airway resistance, single breath N_2 elimination or pulmonary mechanics.) If they grouped the asthmatics and chronic bronchitics together then they could show a significant increase in total lung capacity, functional residual capacity and residual volume. Static compliance was slightly decreased. The authors interpreted their findings as physiologically not meaningful, and that they could have been due to chance. These observations should be repeated at this level as well as at lower levels.

One other study also needs discussion. This is a study by Von Nieding et al (14) in which they exposed healthy subjects either to workplace levels of NO_2 (9.4 mg/m^3 - 5 ppm), O_3 (0.196 mg/m^3 0.1 ppm) and SO_2 (13.1 mg/m^2 - 5 ppm) or NO_2 (0.1 mg/m^3 0.05 ppm), O_3 (0.049 mg/m^3 0.025 ppm) and SO_2 (0.262 mg/m^3 0.1 ppm). Changes in airway resistance and arterial oxygen saturation were observed at the higher levels, whether exposure was to NO_2 or O_3 singly or in combination with or without SO_2. The lower levels produced no effect. Both groups were challenged with 1, 2 and 3% acetylcholine. Acetylcholine, after exposure to the mixture with the higher concentration, produced markedly increased responses over the control values. After exposure to the mixture at the lower levels, there was a slightly increased response after 2% acetylcholine but no increase after 1 or 3% acetylcholine. In view of the lack of an increased response to 3% acetylcholine, the change at 2% does not seem so important. It would have been more relevant if there had been a graded dose response. To propose a hypothesis that the response might be a stepwise function that responded at 2% but not at 3% does not seem to be tenable.

There is no short-term federal standard for NO_2. At this time it does not seem appropriate to set such a standard for one hour at the level of Orehek's study. It would seem to be more appropriate at a level of 0.5-0.8 mg/m^3 (0.266 - 0.425 ppm) as a one-hour average value.

Epidemiologic Studies

There are rather few epidemiologic studies that have looked at the possible effects of NO_2. Those that have, have been compounded by errors in analysis for NO_2 that were discovered later or there had been increased levels of other pollutants along with the increased level of NO_2. Attempts to attribute an effect or a portion of the effect to NO_2 are difficult.

The various studies are summarized in table 2. The studies by Shy et al (9, 10) and Pearlman et al (8) had the problem of a probable error in the Jacob-Hochheisser method of measuring NO_2 as well as the coexistence of elevated particulate levels in conjunction with the elevated NO_2 levels.

The study by Cohen et al (2) was examining such a narrow range of exposure that one might not expect to see much difference.

The studies of Spiezer et al (11, 12) deal with an occupational group - policemen. It may not be appropriate to extrapolate their results to the general public. Also, the areas with the higher levels of NO_2 also had elevated levels of other pollutants. So how much of the effect - if any - could be attributed to NO_2 is not clear.

The Federal standard annual average of 100 mg/m^3 (0.05 ppm) is probably more than adequately protective. Certainly more studies are needed to evaluate the adequacy of this standard.

SO_x and Particulates

These two pollutants will be discussed together because they usually have a common source. In addition, sulfates occur in the particulate phase, either as a mist (H_2SO_4) or as an actual particle as various sulfate salts.

There are two fundamental problems with the category of particulates. One involves the size distribution and the method of collection, the other the chemical composition.

In Europe and Great Britain, particles are collected at relatively low flow rates, on filters that are then read by light reflectance. The system has been calibrated against a standard black coal smoke so that the reflectance units can then be expressed as mg/m^3. This method collects the small size or respirable fraction (<10-7 μm). The conversion applies only to a special coal smoke and cannot be used for oil-fired smoke or for general dusts, etc., that may have a wide range of colors. This method is referred to as the British Standard Smoke method.

In the United States, particulates are sampled by the so-called hi-vol method. In this method, air is drawn through a filter in large amounts (70 - 75 m^3/min) and the amount of dust is measured gravimetrically and referred to as the total suspended particulates (TSP) This method can collect larger particulates that would not be deposited in the lung, although elutriators or other devices can be used to minimize this effect. There is a move to collect the mass respirable fraction (MRP below 10 um) as well as the total suspended particulates. Present U.S. standards are based on TSP. Data are being collected so that hopefully eventually a standard can be set for MRP.

All of these methods-standards imply that equal weight has equal effect. This probably is not so since the chemistry of the particulates needs to be evaluated. Some data have been collected on suspended sulfates, nitrates and a few on the metals. This is an area that needs much more attention. It will be an expensive study but one that needs to be done. The MRP particularly need such analyses as this is the fraction that is more likely to contain the sulfates and also to be deposited deep in the lung. In addition, the hi-vol glass filters tend to collect SO_2 and convert it to sulfate on the filter, thus giving spurious values for suspended sulfates. In due course we should also be able to characterize the type of sulfate, since they do not have equal effect on the respiratory system (15).

Mortality

The effects of acute episodes with markedly high levels of SO_2 and particulates as have occurred in London in 1952 and 1962, the Meuse Vally and Donora are clear evidence that an increased mortality can result. It has been more difficult and controversial to demonstrate an effect at lower levels closer to those usually seen. Buechley et al (16) analyzed mortality data from the New York - New Jersey metropolitan area. They used only one monitoring station to estimate exposure. They did an extensive analysis and controlled for a number of confounding variables. They felt that at about 300 $\mu g/m^3$ SO_2 there was an excess mortality. Particulates, in this case coefficient of haze, did as well as a predictor. Buechley (17) reanalyzed the data later and added additional, more recent years when the levels had fallen. He demonstrated a similar correlation but with a much lower cross-over point. He concluded that SO_2 was acting as a surrogate for some other substance that had not changed as much.

Schimmel and Murawski (41, 42) analyzed the same sort of data using somewhat different methods. They also used a single monitoring station as their indicator of the levels of pollution. They reported that 80% of the effect was due to particulates and 20% to SO_2. In one of their presentations they concluded that SO_2 was relatively unimportant. They too noted no reduction in the mortality with the reduction in pollution.

Lave and Seskin (34) have made extensive analyses relating minimum sulfate and suspended particulate levels to mortality. Their results have received considerable attention. In some cases the results have been accepted and used to calculate the impact on health. Others have been critical of their methodology and conclusion. These are perhaps best summarized by quoting from a group of Biostaticians (43) that reviewed the report of Lave and Seskin.

They were particularly concerned that Lave and Seskin had not investigated as fully as they would wish how well their models fit their data. To determine whether Lave & Seskin's estimates might be severely distorted by outlying values, they undertook a robust analysis and re-estimated the regression given in equation 3.1-1 of Lave and Seskin after some data correction and removal of outliers. The estimates that they obtained for the air pollution regression coefficients are presented in Table 3 along with those of Lave and Seskin. They differ considerably from the values given by Lave and Seskin.

They went on to state:

"Our other area of concern is that the linear model may not be the best-fitting model. Even if it is a good description of current inter-relationships, it may not be suitable for predicting what would occur if the value of the air pollution variables were changed."

"There are several reasons for doubting the suitability. One reason is that a dose-response relationship that can be regarded as linear over a particular range of values may not be linear outside that range. Another reason is that no attention has been paid to the competing risks that cause mortality -- the authors assume that all the other socio-economic factors would remain constant while the air quality changed. A third reason is that association does not necessarily imply causality."

"Our final conclusion is that Lave and Seskin have made a pioneering effort in showing an association between mortality rates and air pollution. The next steps -- assuming a cause and effect relationship and assessing the relative costs and benefits of reducing air pollution cannot, in our opinion, be undertaken with any degree of confidence given the quality and nature of the available data. This conclusion seems to be very close to the Lave and Seskin's own views as expressed in their last chapter, Chapter 11. We believe readers would be well advised to read Chapter 11 before other chapters, because it not only summarizes the rest of the book but also places the work in perspective as a first step in the solution of a complex problem."

It does not seem reasonable to conclude from these studies that SO_x or particulates are having an effect on mortality at the levels usually measured at present. It is not possible to conclude unequivocally that there may not be some effect, but this is probably unlikely.

The report from CHESS, 1970 - 1971 (44) will not be discussed in detail. Their best-estimated values tend to confirm the present Federal standards. Their indictment of sulfates needs much more verification and study. It is unfortunate that the studies were not designed to look at differences between populations as related to differences in the levels of pollution. Instead, they were grouped by area or regions where comparable studies were done.

A number of the studies reported below have come from workers in Great Britain or in Europe where the British Standard Smoke method was used to estimate suspended particulates. Commins and Waller (20) made a comparison of this method with the hi-vol method. Their data have been used to correct the Black Smoke data to total suspended particulates.

Acute or short-term effects

There are a limited number of studies that have looked at the short-term effects on mortality. The effects of a relatively high level on mortality have been well documented. The few studies are summarized in Table 4. The data are spotty and variable.

Jaeger et al (33) exposed 40 mild asthmatics and 40 "normals" to 1300 $\mu g/m^3$ (0.5 ppm) SO_2 for 3 hours. All wore nose clips during the exposure so they were forced to breathe through their mouths. The asthmatics showed a significant decrease in mid-maximal expiratory flow during the exposure. The other tests showed a similar but non-significant trend. Two of the asthmatics and one of the "normals" developed wheezing in the chest during the night after the exposure to SO_2. These were not interpreted as being an asthmatic attack and were considered mild episodes. This study can be criticized in that the battery of pulmonary function tests were not done in the chamber but rather in another laboratory about a four-minute walk away. They did not measure particulates and there was a kerosene heater in the trailer-chamber that could have contributed to the exposure. No organized effort was made to query all the subjects concerning possible symptoms on the night after exposure. This study deserves to be repeated with an improved protocol.

Lawther et al (35) have reported increased symptoms in chronic bronchitics at moderate levels of exposure. Emerson (24) was not able to show any change in pulmonary function in similar types of patients at somewhat higher levels of SO_2. This may have been due to the relatively small number of subjects studied.

Van der Lende et al (45) reported a transitory fall in $FEV_{1.0}$ in a relatively large population at levels below those reported by Emerson. Cohen et al (19) noted a weak association of asthmatic attacks with levels of SO_2 above 200 $\mu g/m^3$ and TSP above 150 $\mu g/m^3$. They reported that temperature acted much more strongly. This was similar to the observation of Emerson.

It does seem that 3-hour exposure to 1300 $\mu g/m^3$ of SO_2 can cause mild symptoms in some sensitive asthmatics. Somewhat lower levels for 24-hour periods in conjunction with levels of TSP above 150 $\mu g/m^3$ cause reversible decreases in $FEV_{1.0}$ and FVC. Since these changes appear to be reversible and their long-range significance is unclear, one could conclude that these changes represent an acceptable change.

Long-term effects

Selected studies on the effects of long-term exposure to SO_2 and particulates are summarized in Table 5. These levels refer to 24-hour annual average values. Here, too, the data reported as black smoke have been converted to TSP equivalents. In Fletcher's study (30) the particulates only had changed markedly; SO_2 levels had not. This was interpreted as emphasizing the role of the particulates. Lawther et al (35) had made a similar observation and conclusion with respect to the short-term exposures. The studies of Sawicki on adults (40) and Lunn et al (36, 27), and Douglas and Waller (23) on children, tend to confirm the levels of particulates.

Chapman et al (18) have also studied children. Their study focused primarily on particulates. They shifted their instrument that they used to measure the pulmonary function without reporting any tests to show comparability. Also, there were differences in the socio-economic status that could have accounted for the differences seen. A further problem lies in the tacit assumption that equal weight of particulates means an equal effect. This is probably not true. There is a great need to develop more information on the particle-size distributions and the chemical characteristics of the various particle-size groups.

Hammer et al (31) did very extensive analyses to control for a variety of confounding factors in two groups of children in Long Island and New York City. They concluded that there was an excess of lower respiratory disease mortality when SO_2 levels were 175 - 250 $\mu g/m^3$ (0.067 - 0.096 ppm), TSP above 115 $\mu g/m^3$ and suspended sulfates of 13 - 14 $\mu g/m^3$. As with all of these studies, the exposures are best estimates from a single monitoring station. Their suggested levels are consistent with observations of others. No analyses were done to try to separate out the effects of SO_2, particulates or sulfates. Certainly their data are not sufficient to use to set a standard for sulfates.

The last three studies represent a 12-year series of follow-up studies, on a community in New Hampshire (26, 28, 29), where the main source of pollution is a pulp mill. The same sample was followed and as the levels of pollution fell an effect level appeared at about 130 $\mu g/m^3$ TSP. The total sulfur as measured by the lead peroxide candle was low and underwent some fluctuations reflecting the closing of the sulfite operation in 1963 and the expansion of the Kraft process in subsequent years.

The results from the initial study have been compared with comparable data from Great Britain (38) and with a cleaner community in British Columbia (27). This comparison indicated a sort of a dose-response effect in that more polluted communities in Great Britain had more symptoms than in Berlin, N.H. and the cleaner community had better pulmonary function. Thus, the observation from the community with a pulp mill may well be applicable to other communities. It would have been good to have had information as to the chemical composition of the particulates, as they undoubtedly were different in the three communities.

Sulfates

The Chess (44) studies have reawakened interest in sulfates as possibly being more important than SO_2. A fundamental problem with sulfates is that it is probably the cation that is more important. There appears to be a gradation of effects in animals (15) with sulfuric acid being the most irritating and sodium sulfate being relatively innocuous. We are not yet able to characterize the type of sulfate. Thus, a general standard for soluble sulfates does not seem to be warranted given the range of irritancy for the different sulfate compounds.

An assessment of the effects of microparticulate sulfates on human health has been prepared (25). It is appropriate to comment on some of the studies reported there as well as some more recent work.

Dohan (21) and Dohan and Taylor (22) looked at the prevalence of respiratory disease in a number of cities related to levels of suspended particulate sulfates. They showed an increased prevalence with rising levels of sulfates. This was exacerbated during a flu epidemic. Their threefold rise in suspended sulfates was accompanied by an increase in total suspended particulates from something over 100 $\mu g/m^3$ to 188 $\mu g/m^3$. They concluded that sulfates were the active component. However, they had not controlled for the various confounding variables.

A similar study was undertaken by Ipsen et al (32) in which much more detailed and sophisticated analyses were done to control for the auto-correlation between pollutants and season of the year. When they controlled for the auto-correlation, they were not able to show that the levels of pollution were having any effect on respiratory disease. Their studies emphasize the need to control for such phenomena in order not to be tempted to make incorrect conclusions.

Sackner et al (39) have been exposing animals and human beings to submicronic sulfuric acid mist. Human beings have been exposed up to 1000 $\mu g/m^3$ for 10 minutes without demonstrating any effects on a variety of tests of pulmonary function. This has included some asthmatic children. These results are encouraging as to the toxicity of sulfuric acid mist but they need to be replicated and also for longer periods of exposure. Exercise should also be added to the protocol.

Miscellaneous

Some of the combustion products that are emitted from coal-fired plants have carcinogenic capabilities. These materials, like the variety of metals that can also be emitted, are probably in too low a concentration to be having an effect. Careful studies to evaluate this potential problem still need to be done to have better quantification of the risk and also to determine whether there may be synergism with such exposure and occupational exposure as well as with cigarette smoking.

No discussion is planned of the role of sulfur oxides and oxides of nitrogen to the formation of acid rains. These may well turn out to be the controlling factor in control of emmissions.

Summary:

The most significant potential contributions to air pollution from fossil-fuel-fired power plants are NO_2, sulfur oxides and particulates. Their possible effects on health are discussed.

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Table 1

Selected Summary of Results of Chamber Studies (short-term) on Human Beings Exposed to NO_2

Concentration mg/m^3	 (ppm)	Duration of Exposure	Effects	References
4.5	(2.5)	2 hrs.	Incr. R_{aw} normals to incr. to Ach response	1
<2.82	(<1.5)	0.25 hr.	No change R_{aw} health subjects	13
1.316-1.880 plus other smog components	(0.7-1.0)	Not specified until pulse 150/min on exercise	No significant change in reaction time or cardiorespiratory work efficiency in healthy subjects	4
1.166	(0.62)	2 hrs. exercise 0.2 - 1 hr. ventilation incr. 4 x	No change; no diff. exercise healthy subjects	5
0.940	(0.5)	2 hrs.	13 asthmatics, 7 chr. br., 10 health normals; 7/13 asthmatics: slight burning eyes (2) headache (1) and chest tightness exercise (4); no significant change in pulm. function unless comb. patient groups; changes very slight	6
0.564 added to O_3 exposure	(0.3)	2 hrs.	No increased response in "reactive" subjects	3
0.200	(0.11)	1 hr.	3/20 asthma inc. R_{aw} 13/20 increased sensitivity Carbacol; no symptoms	7
0.10 (also ozone 0.05, SO_2 0.26)	(0.05)	2 hrs.	No effect gas exchange or R_{aw}; 2% Ach incr. R_{aw} 1% & 3% no effect; healthy volunteers	14

Table 2

Selected Summary of Epidemiologic Studies on NO_2

Concentration mg/m^3 Mean	(ppm) 90th Percentile	Effects	References
0.15 - 0.28* (0.08 - 0.15)	0.28 - 0.94* (0.15 - 0.54)	Slight increase in respiratory symptoms and slight decrease in pulmonary function	9, 10
Same		Slight increase in bronchitis morbidity - No effect on croup, pneumonia or hospitalizations	8
0.096 vs 0.043 (0.051 vs 0.023)	0.188 vs 0.113 (0.10 vs 0.06)	No effect healthy non-smoking adults	2
0.103 (0.055	Maximum 0.263 - 0.654 (0.140 - 0.30	Questionable small increase in respiratory symptoms. No difference in pulmonary function; Policemen.	11, 12

* Data from Jacob-Hochheiser method. Later shown to be in error (low); values probably should be higher.

Table 3

Comparison of Regression Coefficients (b - values) Calculated by Lave and Seskin (34) and Thibodeau et al (43)

	Sulfates		Particulates	
	Lave & Seskin	Thibodeau, et al	Lave & Seskin	Thibodeau, et al
Minimum	0.473	0.08	0.199	0.57
Mean	0.173	0.26	0.303	0.01
Maximum	0.028	0.08	-0.018	0.01

Table 4

Selected Summary of Effects of Short-Term Exposures to SO_2

and/or Particulates - 24-hr Level Unless Otherwise Specified

SO_2		Suspended Particulates	Effects	Ref.
$\mu g/m^3$	(ppm)	$\mu g/m^3$		
1300	(0.5) 3 hrs	--	Chamber exposure, mouth breathing, Resting, Slt decr. MMEF asthmatics; 2/40 asthmatics wheezed in night; 1/40 "normals" wheezed in night	33
722	(0.276)	350[a]	No change in pulmonary function in patients w/ chronic bronchitis	24
300-200	(0.114 - 0.070)	230[a]	Reversible Decreased $FEV_{1.0}$	45
250	(0.095)	350[a]	Increased respiratory symptoms in patients w/ chronic bronchitis	35
200	(0.076)	150	Weak association asthmatic attacks	19

[a] Corrected from orginal data to TSP equivalents (20).

Table 5

Selected Summary of Effects of Long-Term Exposures to SO_2 and Particulates; Levels Refer to 24-hour Annual Averages

SO_2		Suspended Particulates	Effects	Refs.
$\mu g/m^3$	(ppm)			
250	(0.095)	250[a]	Increased phlegm production	30
130	(0.05)	240[a]	Increased respiratory disease	40
120	(0.046)	200[a]	Increased respiratory illness and decreased pulmonary function	36,37
120	(0.046)	230	Increased lower respiratory illness	23
23	(0.009)	110	Decreased $FEV_{0.75}$	18
250-175	(0.096-0.067)	115	Increased lower respiratory disease and morbidity	31
55	(0.021)[b]	180	Increased respiratory symptoms Decreased pulmonary function	26,27
37	(0.014)[b]	131	No effect	28
66	(0.025)[b]	80	No effect	29

[a] Corrected from original data to TSP equivalents (20)

[b] Equivalents calculated from lead peroxide data

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HUMAN EXPOSURES TO WATERBORNE POLLUTANTS FROM COAL-FIRED STEAM ELECTRIC POWERPLANTS

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The focus of this paper is the evaluation of the potential impact on human health from waterborne chemical pollutants emitted principally at coal-fired steam electric powerplant sites. Coal is the primary fossil fuel used for electric power generation in the Ohio River Basin, which is the major reason for its emphasis in this paper. Nevertheless, such plants have substantial emissions in common with other fossil-fueled and nuclear powerplants, and thus, where applicable such as for cooling water effluents, some of the total impacts of these common emissions will be considered when assessing total wastewater flows. Although many of the chemicals which are constituents of the coal itself will also be emitted as waterborne wastes due to coal mining, preparation, and transportation, the possible impacts of these activities will not be considered specifically.

It is not the intention of this paper to exhaustively present the full range of chemical pollutants emitted by the water route at these powerplants. Such information can be found in numerous articles and reports. Rather, using a few specific examples, an attempt will be made to assess some of the likely human exposures that may occur, principally through the drinking water route, once these chemicals are emitted to surface receiving waters, primarily rivers, and assuming that they act as conservative pollutants. In this context, and perhaps somewhat unusually, the term conservative will signify: first, that they do not degrade, which certainly applies to trace elements in stable oxidation states; that they are not released by the natural water system, such as by precipitation or volatilization; and finally that they are not affected by municipal water treatment processes. It is safe to say that it is unlikely that one could find any water pollutant that meets all of these conditions, but, as a preliminary worst-case analysis, this is certainly a conservative set of conditions in two senses of the term.

After initially describing briefly the kinds of sources of wastewaters associated with fossil-fueled steam electric powerplants, two broad classes of chemicals will be considered: trace elements and organics. Some of their uses and origins at these sites will be reviewed, along with some of their quantities emitted, and possible impacts on surface water quality, and ultimately, possible human exposures. Although it is well known that such human waterborne exposures can be considerably amplified, such as for mercury, via concentration and accumulation in the aquatic food chain, because of limited quantitative information this possible route will not be evaluated. Nevertheless, any conclusions based on assessments of waterborne exposures that do not consider such additional routes must be regarded as incomplete.

There are a variety of wastewater streams associated with powerplants and these are shown schematically in Figure 1 (1). They vary considerably in flow volumes, ranging from as much as 1 MGD/Mw typically for once-through cooling, to just a few gallons per day per megawatt for some cleaning operations, with recirculating cooling water blowdown and once-through ash sluicing systems being intermediate in flow at several thousand GPM/Mw. The composition of the intermittent smaller flows of run-off from coal piles, as well as that of the bottom and fly-ash wastewaters, is largely determined by coal composition, while many other wastes, such as cooling water, wastewater from boiler water treatment and boiler condensate blowdown are strongly influenced by added chemicals. The categories of these wastewaters by relative volumes and rainfall runoff influence are listed in Table 1.

A large variety of organic and inorganic chemicals are utilized for water treatment, cleaning, and the control of corrosion and biofouling of cooling water systems, many of which are shown in Table 2. These additives, along with coal constituents can and do appear in these cooling water and wastewater effluents. Because of the very large volumes from once-through cooling, which is estimated to be used currently in about two-thirds of the steam electric powerplants in the U.S., a portion of this paper will consider the potential impact of the formation of chloro-organics in such systems due to the wide use of chlorine to reduce bio-fouling.

Many trace elements, including heavy metals, can appear in virtually every wastewater and cooling water effluent stream of powerplants at concentrations higher than those in the source water. Such chemicals can originate from the coal itself, from corrosion products at the plant, and from chemicals used for such purposes as cleaning agents, corrosion inhibitors, such as zinc and chromium, and water and wastewater treatment chemicals, such as alum,used as a coagulant in water treatment,and contaminants of lime. In some cases there is the potential for reducing their emissions by using alternative chemicals, such as the possible substitution of molybdate as a corrosion inhibitor instead of chromium or zinc. However, when the trace element originates from the coal itself, it may be possible to reduce its emission only by modification of the wastewater flow systems or treatment of their effluents. Thus, for example, the diversion of run-off from a coal storage pile to an ash lagoon can reduce its impact.

Unlike the trace organics, considerable data are available regarding the concentrations of trace elements emitted in wastewater flow streams at powerplants. To obtain a perspective on the possible impact of a few such metals in wastewaters of powerplants, Table 3 presents estimates of their daily quantities discharged compared to those from all other industrial sources. It is clear that for these four metals, powerplants constitute a significant, if not substantial source. In the case of chromium this 1973 estimated discharge rate was perhaps 0.5 percent of all chromium actually used by industry in the U.S. Similarly, it was recently estimated by the E.P.A. that during the period 1975 to 1990 electric utilities would discharge about 50 percent of all the lead emitted to water. It is, therefore, apparent that there is a need to assess the possible impacts of the emissions of these and other trace elements as possible agents of waterborne disease.

Some typical concentrations of zinc and chromium applied as corrosion inhibitors in cooling towers are shown in Table 4, along with information about a few other chemicals. It should be emphasized that these typical concentrations in the circulating water are in units of mg/l, and are quite substantial should they ultimately appear in a receiving stream and remain relatively undiluted. However, the volumes of such blowdown flows are much smaller than those from once-through cooling, and do in fact often receive considerable dilution by receiving waters. Where there may be reason for concern, in spite of the high dilution, is the possible precipitation and incorporation of these and other heavy metals into sediments, which can then constitute a major sink and act as a substantial source in the aquatic food chain, such as for shellfish. Thus, where an analysis indicates that the average impact of such effluents on receiving waters and ultimately drinking water is small, other warerborne routes of exposure should be considered before a hazard is judged to be unlikely.

To obtain an additional perspective on some of the wastewater streams from a coal-fired powerplant, Table 5 shows some typical flows in gallons per day per megawatt (GPD/Mw) from several plant operations. It is apparent that there is a considerable range of flows as discussed earlier, as well as concentrations of trace and macro constitutents within them. The flows shown there are substantially lower than the typical value of 1 MGD/Mw for once-through cooling, which is also much larger than, perhaps typically , 10,000 GD/Mw for blowdown from a recirculating cooling water system. However, the latter flow and that from a once-through ash pond overflow are of the same order of magnitude.

In all of these wastewater streams one can find a full range of trace elements, and these have been studied extensively. Some examples of their concentrations in coal pile leachates are shown in Table 6 (2). It is apparent that the concentrations of many of these chemicals can be quite high compared to typical concentrations in the Allegheny River, as well as drinking water criterion limits. However, because of the relatively low average volumes of these leachates, they are unlikely to constitute a hazard except for a local situation, such as due to non-dilution or contamination by infiltration into a water supply aquifer. Another possible route of impact on water quality of these trace elements is by the ultimate settling of particulates from stack emissions at powerplant sites, and their subsequent leaching into surface or ground waters. No such direct assessment of this route will be presented here, but it is incorporated into the following regional assessment.

It is useful to consider a possible future impact of trace element emissions from increased power utilization in a specific region. One such analysis by the Regional Studies Program of the Argonne National Laboratory did such a projection to the year 2020 for several areas, including four counties in the Illinois River basin in Illinois (3). As part of the National Coal Utilization Assessment they projected the emissions of trace elements in a combined high coal electric and accelerated synfuel scenario. This region was chosen as a case study because of the large amounts of accessible coal reserves and the potential for increased coal-fired power generation and synthetic fuel production. It should be emphasized that their projected emissions include those related to several phases of coal utilization, including the possible impact of urbanization. Also particulate atmospheric emissions that are settleable were included. In this case they assumed that 50 percent of the deposited atmospheric trace elements are transported to the river via surface run-off. Although it is apparent that their analysis encompasses much more than impacts of the coal-fired electric powerplant itself, to the extent that it assesses the full range of sources, it can indicate whether indeed there may be cause for concern about operations at the powerplant site.

As an example of their approach we shall examine their analysis for Jersey county, one of four they considered in the Illinois River basin. Table 7 presents their data for eight trace elements that have potential impacts on human health. In each case average concentrations in the river in the absence of increased coal utilization are shown, as well as some current drinking water standards or criterion limits. Incidentally, the average values for arsenic and cadmium in the river exceed these limits. However, water treatment will often reduce concentrations of trace elements. Table 7 indicates that there are substantial projected increments under average flow conditions due to the 2020 scenario, at least for some of the trace elements, including selenium, mercury and beryllium. And, of course, under the 7-day 10-year low flow condition, there is a substantially greater impact, by a factor of about 8. In this case the increments compared to background concentrations become important for the other elements as well. It should, however, be emphasized that where the human effects of these elements are chronic, the short period low-flow higher exposure becomes of lesser concern. Finally, the last column considers the relative body burdens due to drinking water intake under low flow conditions compared to typical dietary sources. For mercury and lead the water constitutes a substantial source, but again these would be reduced by a factor of about 8 under average flow conditions. It should also be noted that their assessment also indicates that the air contributions to these body burdens are much lower than those of water. One can thus conclude that, to the extent that the assumptions are valid, it is unlikely that there is a substantial impact from most of these trace elements on the human health of the general population in the year 2020 scenario in Jersey County, Illinois. Where the analysis indicates that an element like mercury may be of concern, there may be need to refine the assumptions and examine further the extent to which, for example, the mercury may be behaving as a conservative pollutant in the river and water treatment systems.

One can at least make several preliminary judgements and conclusions about the possible waterborne route impact on human health from trace elements arising from steam electric coal-fired powerplants. There can be substantial quantities of such trace elements emitted, even when compared to those associated with other anthropogenic activities. Some of these emissions are due to the use of chemicals at the plant and can be limited by the judicious use of alternative chemicals. However, a large number of trace elements are present in coal and will necessarily appear in wastewater streams, although their impact can be reduced. These trace elements are naturally present in the hydrosphere and in many cases and at most times the incremental loads from powerplants are very small. However, for some elements, such as mercury, selenium, arsenic, chromium, and lead, the added waterborne load could be substantial compared to background concentrations. In particular, situations could be encountered where there are localized impacts, such as due to low dilution, groundwater contamination, or concentration in sediments. In general, however, the concentrations of trace elements in water are such that potable water sources constitute a relatively small fraction of the contributions to human body burdens compared to dietary sources. Where there is a potential increase from powerplant emissions, it is unlikely that they will constitute a danger of acute human toxicity. However, there is a need to fully and continually assess their potential chronic effects as such powerplants become regionally concentrated and their emissions become highly localized.

Next we shall address the organic chemicals that can appear in wastewater effluents at coal-fired steam electric powerplants, and these can be attributed to four basic sources: those present in the raw source water, either of natural or anthropogenic origin; those orginating from the coal itself, ultimately appearing, for example, in ash pond effluents or run-off from coal storage piles; chemical additives, such as corrosion inhibitors and cleaning and dispersing agents; and products from the reactions of chemicals in the water and wastewater systems, such as those from chlorine, added to cooling water and then reacting either with natural or synthetic organics already present in or added to the water. Serious concern about such chlorination reactions has developed in relation to the formation of trihalomethanes in drinking water disinfection, but many other products, depending on the nature of the organic precursor, can form as well. The E.P.A. list of 129 priority pollutant chemicals consists mostly of organic chemicals and includes several of these trihalomethanes that can form in water and wastewater chlorination. All of priority pollutants are now being addressed for possible control in the proposed revision of steam electric powerplant effluent limitations guidelines (4).

Only sparse data seem to be available on the nature and concentration of organics actually present in wastewater or cooling water from these powerplants. A compilation of some such measurements reported recently by the E.P.A. for several powerplants is shown in Table 8 (4). In addition to the organics listed there, others were detected frequently, such as unchlorinated phenol at concentrations of a few μg/l in cooling tower blowdown, several phthalates, and several polycyclic aromatic hydrocarbons, which as a class are frequently associated with coal process wastes and contain some highly potent animal carcinogens.

Of the organics listed in Table 8, benzene can be categorized as a suspected human carcinogen and chloroform as an animal carcinogen. For almost all the other listed chemicals there are either very little or no available data on chronic toxicity, although acute human toxic effects are known at much higher levels of exposure. Several of these chemicals have, however, unpleasant taste and odor characteristics, and at the concentrations shown can impart unpleasant taste to fish or similarly taint drinking water. As a class halogenated organics are responsible for adverse biological reactions and are the subject of regulatory initiatives.

Although the concentrations shown in Table 8 are in the μg/l range, in most cases they typically are at least comparable to and often substantially larger than the highest concentrations found in drinking water (5). Whether they will result ultimately in significant waterborne human exposure depends on dilution of the wastewater streams, their ability to concentrate in the food chain, their movement and fate in natural waters, and their ability to survive drinking water treatment.

In an attempt to assess the possible impact of the reactions of chlorine with organic precursors in powerplant cooling water, Jolley and co-workers in the laboratory exposed untreated cooling water sources to the chlorine doses they would receive typically in practice, using radiotracer chlorine-36 and assessing its incorporation into the gross organic load, as well as measuring some specific chloro-organics that were formed (6). Chlorine is frequently used in such cooling water to prevent bio-fouling of the cooling water system. The tentative identification and quantification of several of the compounds found is shown in Table 9, along with comparable results for a chlorinated secondary sewage effluent. There is considerable concern about the possible adverse effect of chlorine residuals and such chloro-organic compounds on aquatic biota, and it is interesting to note that the concentrations in the chlorinated secondary sewage effluent were comparable to those in the other chlorinated waters. Many of these compounds are known to kill or otherwise affect fish or zooplankton. For example, it has been reported that 5-chlorouracil and 4-chlororesorcinol can significantly lower the hatching success of carp eggs at concentrations as low as 1 μg/l (7). Nevertheless, aside from organoleptic characteristics of the chlorophenols shown in Table 9, any possible adverse chronic human effects at these concentrations are generally not known. However, they are of interest as an indication that a wide variety of chloro-organics can be formed in chlorinated cooling waters at powerplants.

In these chlorination studies it was observed that the range of incorporation of chlorine into chloro-organic matter was 0.5 to 3 percent of the applied chlorine dose of approximately 3 mg/l, which is typical of that used in chlorinating secondary sewage effluents and in once-through cooling water systems at powerplants, although the latter usually receive intermittent doses. From these results one can infer that there was an incorporation of a range 15 to 90 μg/l of chlorine into organic matter in the cooling water. The actual quantity of organic matter thus ending up as chloro-organics would, of course, be highly dependent on their nature. Taking chlorobenzoic acid as a model, this range of chlorine incorporation would generate 65 to 390 μg/l of total chloro-organics. If, however, all the chlorine were incorporated into the highly chlorinated chloroform, this would generate 17 to 101 μg/l.

In order to evaluate the possible ultimate human exposures to these chlororganics from powerplant effluents it is useful to first assess their potential impacts on surface water quality, particularly in comparison to the loads generated by chlorinated municipal sewage. This is addressed in Table 10 for four segments of the Ohio River and two of its major tributaries in the greater Pittsburgh area. The quantities of power generated along these river segments are shown, along with the estimated or known cooling water effluent flows (including both once-through and other types), chlorinated sewage effluent flows, and average and recent minimum river flows. It is apparent that, except for the Ohio River from Pittsburgh to the Beaver River, the ratio of cooling water effluent flow to that from chlorinated sewage is quite high. On the average for the region, this ratio is about 8 to 1. It is interesting to note that in the lower Allegheny and Monongahela Rivers the cooling water effluent flows are about 40 and 60 percent, respectively, compared to recent minimum river low flows, although they are substantially smaller than the average river flows. It is thus apparent that the chlorinated organics in these cooling water effluents could appear in the surface receiving waters relatively undiluted at times. Also, because the doses of applied chlorine are comparable for the cooling water and secondary sewage effluents, it is reasonable that they may be generating comparable loads of chloro-organics when their relative volumes and frequency of chlorination are considered, at least in the Pittsburgh area described in Table 10.

One can make several generalizations and conclusions from these various sources of information regarding the possible impacts of organics from steam electric powerplants. Measurable concentrations in the region of 1 to 100 μg/l of a large variety of organic chemicals can be found in the wastewaters of such powerplants, although the available data are quite limited. In addition to the organics from the coal itself and those added in the plant, such as for water treatment, corrosion inhibition and cleaning, chlorination of cooling water can result in quantities of chloro-organics comparable to those generated by the chlorination of municipal sewage, and in some areas, possibly even much greater quantities. The possible impacts on water quality will be highly site-specific, and under some low flow conditions the dilutions of these cooling water effluents may be very small. Although a few of the identified organics are likely to be human carcinogens, most occur at concentrations well below known acute toxic levels, and at the same time little or no information is available regarding their chronic human effects. In addition to human exposure via drinking water, they could also affect human health by concentration in the aquatic food chain. However, it is a reasonable judgement that there is no need for alarm about their possible impacts. At the same time it would be highly desirable to obtain additional information about the full range of organics in these effluents and their likely concentrations. Such evaluations could then be put in the perspective of the variety of information now being developed about the possible human health effects of such compounds in water, and a better assessment then made as to the contributions and impacts from powerplants.

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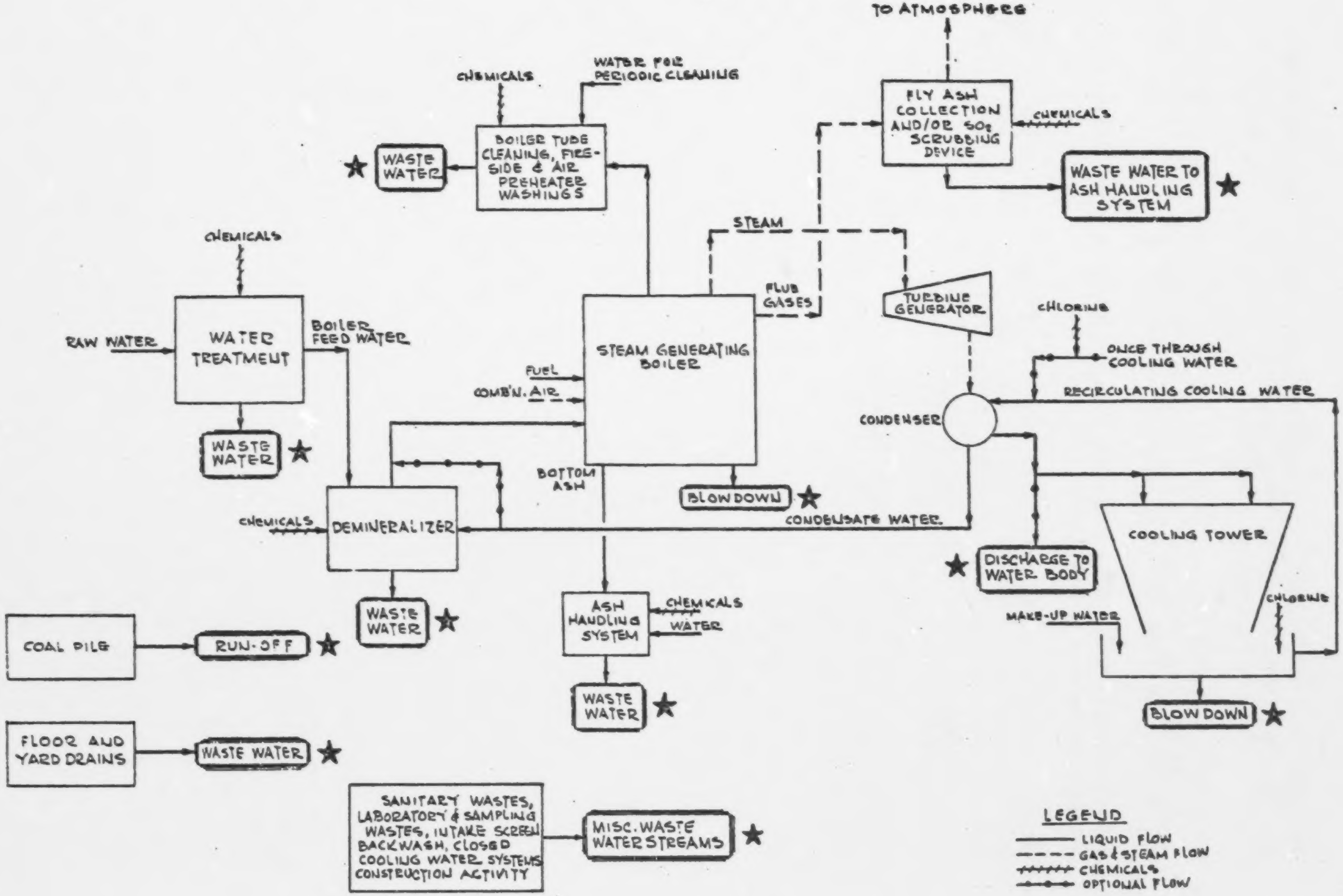

FIGURE 1. TYPICAL WATER AND WASTEWATER FLOW DIAGRAM FOR FOSSIL-FUELED STEAM ELECTRIC POWERPLANT (FROM EPA 440/1-74 029-a)

TABLE 1 . CLASSIFICATION OF VARIOUS WASTEWATER SOURCES FROM STEAM ELECTRIC POWER PLANTS (FROM EPA 440/1-74 029-a)

Class	Source
High Volume	Nonrecirculating main condenser cooling water
Intermediate Volume	Nonrecirculating house service water Blowdown from recirculating main cooling water system Nonrecirculating ash sluicing systems Nonrecirculating wet-scrubber air pollution control systems
Low Volume	Clarifier water treatment Softening water treatment Evaporator water treatment Ion exchange water treatment Reverse osmosis water treatment Condensate treatment Boiler blowdown Boiler tube cleaning Boiler fireside cleaning Air preheater cleaning Stack cleaning Miscellaneous equipment cleaning Recirculating ash sluicing systems Recirculating wet-scrubber air pollution control systems Intake screen backwash Laboratory and sampling streams Cooling tower basin cleaning Rad wastes Sanitary system Recirculating house service water Floor drainage Miscellaneous streams
Rainfall Runoff	Coal pile drainage Yard and roof drainage Construction activities

TABLE 2 . CHEMICALS USED IN STEAM ELECTRIC POWER PLANTS (FROM EPA 440/1-74 029-a)

Use	Chemical
Coagulant in clarification water treatment	Aluminum sulfate Sodium aluminate Ferrous sulfate Ferric chloride Calcium carbonate
Regeneration of ion exchange water treatment	Sulfuric acid Caustic soda Hydrochloric acid Common salt Soda ash Ammonium hydroxide
Lime soda softening water treatment	Soda ash Lime Activated magnesia Ferric coagulate Dolomitic lime
Corrosion inhibition or scale prevention in boilers	Disodium phosphate Trisodium phosphate Sodium nitrate
pH control in boilers	Ammonia Cyclohexylamine
Sludge conditioning	Tannins Lignins Chelates such as EDTA,NTA
Oxygen scavengers in boilers	Hydrazine Morphaline
Boiler cleaning	Hydrochloric acid Citric acid Formic acid Hydroxyacetic acid Potassium bromate Phosphates Thiourea Hydrazine Ammonium hydroxide Sodium hydroxide Sodium carbonate Nitrates
Regenerants of ion exchange for condensate treatment	Caustic soda Sulfuric acid Ammonex
Corrosion inhibition or scale prevention in cooling towers	Organic phosphates Sodium phosphate Chromates Zinc salts Synthetic organics
Biocides in cooling towers	Chlorine Hydrochlorous acid Sodium hypochlorite Calcium hypochlorite Organic chromates Organic zinc compounds Chlorophenates Thiocyanates Organic sulfurs
pH control in cooling towers	Sulfuric acid Hydrochloric acid
Dispersing agents in cooling towers	Lignins Tannins Polyacrylonitrile Polyacrylamide Polyacrylic acids Polyacrylic acid salts
Biocides in condenser cooling water systems	Chlorine Hypochlorites
Additives to house service water systems	Chlorine Chromates Caustic soda Borates Nitrates
Additives to primary coolant in nuclear units	Boric acid Lithium hydroxide Hydrazine
Numerous uses	Numerous proprietary chemicals

TABLE 3. TOTAL METALS DISCHARGED FROM POWERPLANTS IN THE U.S. (1973) COMPARED TO OTHER INDUSTRIAL SOURCES, INCLUDING COOLING WATER DISCHARGES (FROM EPA 440/1-74 029-a)

Pollutant	Discharges by Major Steam Electric Powerplants, lb/day	Percentage of All Major Dischargers
Chromium	15,365	50
Copper	2,739	14
Iron	20,683	10
Zinc	20,099	21
Total	58,886	14

TABLE 4. WASTE DISPOSAL CHARACTERISTICS (WITH CONCENTRATIONS IN mg/l) OF TYPICAL COOLING TOWER INHIBITOR SYSTEMS (FROM EPA 440/1-74 029-a)

Inhibitor System	Concentration in recirculating water
Chromate only	200-500 as CrO_4
Zinc	8-35 as Zn
Chromate	17-65 as CrO_4
Chromate	10-15 as CrO_4
Phosphate	30-45 as PO_4
Zinc	8-35 as Zn
Phosphate	15-60 as PO_4
Zinc	8-35 as Zn
Phosphate	15-60 as PO_4
Phosphate	15-60 as PO_4
Organic	3-10 as organic
Organic only	100-200 as organic 10 est. as BOD 100 est. as COD 50 est. as CCl_4 extract 5 est. as MBAS
Organic Biocide	30 as chlorophenol 5 as sulfone 1 as thiocyanate

TABLE 5 . TYPICAL CHEMICAL WASTES FROM A COAL-FIRED POWERPLANT
(FROM EPA 440/1-74 029-a)

Waste Stream	Flow, GPD/Mw	Typical Concentrations of Major Pollutants ,mg/l				
		TSS	Iron	Copper	Sulfate	Hardness
Ion exchange	88	46	-	-	2085	-
Boiler blowdown	52	25	-	-	-	-
Boiler cleaning	0.25	127	2100	380	-	520
Boiler fireside cleaning	4.44	582	142	-	1650	4661
Air preheater cleaning	11.7	1882	1610	-	1130	3700
Miscellaneous cleaning	1.11	1000	-	-	-	-
Laboratory operations	10	100	-	-	-	-
Floor drains	30	100	-	-	-	-
Recirculating bottom ash sluicing blowdown	400	1000	-	-	-	-
Ash pond overflow (once-through fly ash)	5000	60	-	-	510	244
Coal pile drainage	60	864	-	-	6880	1025

TABLE 6 . COMPARISONS OF SOME TRACE ELEMENT CONCENTRATIONS IN COAL PILE LEACHATES WITH THOSE IN THE ALLEGHENY RIVER AND HEALTH AND OTHER DRINKING WATER CRITERION LIMITS (µg/1)

Element	Leachate, Plant 1[a]	Leachate, Plant 2[a]	1966 mean, Allegheny R.	EPA[b] criterion limit
Cu	400 - 1,000	10 - 500	30	1,000
Zn	2,000 - 16,000	1,000 - 4,000	90	5,000
Ni	700 - 5,000	200 - 500	20	-
Cr	< 5 - 10	< 5 - 10	5	50
Hg	< 0.2 - 3	3 - 7	-	2
As	5 - 600	6 - 50	60	50
Se	< 1 - 30	< 1 - 1	-	10
Be	30 - 70	< 10 - 30	0.1	0.2[c]

[a] From D.B. Cox, T.Y.J. Chu, and R.J. Ruane, in Proceedings of NCA/BCR Coal Conference, Louisville, Ky., October 1977.

[b] U.S.E.P.A. Interim Primary Drinking Water Regulations and Proposed Secondary Regulations.

[c] U.S.S.R. Drinking Water Standards

TABLE 7. YEAR 2020 PROJECTION OF TRACE ELEMENT IMPACT FROM A COMBINED HIGH COAL ELECTRIC AND ACCELERATED SYNFUEL SCENARIO: WATER QUALITY IN ILLINOIS RIVER (JERSEY COUNTY) AND BODY BURDEN*

	Concentration - µg/l				
		Year 2020 coal utilization increment			
Trace element	River average	Average Flow	7-day 10-year low flow	Drinking water criterion	Water‡ vs. diet body burden
As	44	0.6	5	10	0.23
Be	0.10	0.13	1.0	0.2	0.11
Cd	12.7	0.7	5.7	10	0.03
Cr	7.4	1.1	8.4	50	0.32
Hg	0.0	0.4	2.9	2	2.3
Ni	12.9	2.5	19.8	1000	0.08
Pb	28.3	2.9	22.7	50	0.73
Se	0.01	0.5	4.1	10	0.02

*Adapted from An Integrated Assessment of Increased Coal Use in the Midwest: Impacts and Constraints, ANL/AA-11 (Draft), Argonne National Laboratory, October 1977.

‡Comparing projected 7-day 10-year low flow concentration in river (assuming same in drinking water) with typical diet intake.

TABLE 8 . SOME REPORTED CONCENTRATIONS (μg/l) OF ORGANIC CHEMICALS IN WASTEWATERS FROM STEAM ELECTRIC POWERPLANTS*

Chemical	Once-thru cooling	Cooling tower blowdown	Ash pond effluent
Benzene	-	2 - 45	1 - 2
Toluene	-	24	4
2,4-dinitrophenol	-	-	50
Dichlorobenzene (isomers)	-	20	35 - 64
2,4-dichlorophenol	-	-	83
2.4.6-trichlorophenol	-	35	-
1,2-dichloroethane	44	-	-
1,1-dichloroethylene	16	-	-
1,2-dichloroethylene	11	-	-
1,1,1-trichloroethane	12	-	27
Tetrachloroethylene	78	-	-
Chloroform	-	2 - 26	-
Bromoform	-	13 - 154	-
Chlorodibromomethane	-	59	-

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*From Technical Report for Revision of Steam Electric Effluent Limitation Guidelines (Draft), U.S. Environmental Protection Agency, September 1978.

TABLE 9 . TENTATIVE IDENTIFICATION AND CONCENTRATIONS (μg/l) OF CHLORO-ORGANICS IN LABORATORY-CHLORINATED SAMPLES OF SEWAGE EFFLUENT AND STEAM ELECTRIC POWERPLANT COOLING WATER INFLUENT[a]

	Sewage Effluent	Watts Bar Lake Sample	Mississippi River Sample
Nucleoside			
5-chlorouridine	1.7	0.6	7
Purine			
8-chlorocaffeine	1.7	1.1	6
6-chloro-2-aminopurine	0.9	1.0	3
8-chloroxanthine	1.5	3	-
Pyrimidine			
5-chlorouracil	4	0.6	3
Aromatic acid			
2-chlorobenzoic acid	0.3	1.1	10
3-chlorobenzoic acid	0.6	0.2	8
4-chlorobenzoic acid	1.1	0.3	8
3-chloro-4-hydroxybenzoic acid	1.3	0.8	3
4-chloromandelic acid	1.1	1.8	6
4-chlorophenylacetic acid	0.4	3	20
5-chlorosalicylic acid	0.2	3	18
Phenol			
4-chloro-3-methylphenol	1.5	0.2	0.7
2-chlorophenol	1.7	0.2	4
3-chlorophenol	0.5	0.2	6
4-chlorophenol	0.7	0.2	2
4-chlororesorcinol	1.2	0.5	7

[a] From R.L. Jolley, G. Jones, W.W. Pitt, and J.E. Thompson, "Determination of chlorination effects on organic constituents in natural and process waters using high-pressure liquid chromatography," in Identification and Analysis of Organic Pollutants in Water, L.H. Keith (Ed.), Ann Arbor Science, 1976.

TABLE 10 . COMPARISON OF ESTIMATED STEAM ELECTRIC POWER COOLING WATER AND CHLORINATED MUNICIPAL SEWAGE EFFLUENT FLOWS WITH THOSE OF THEIR RECEIVING WATERS IN MAJOR RIVERS OF SOUTHWESTERN PENNSYLVANIA

River segment	Power generated	Cooling water effluent	sewage effluent	Avge. river flow	Recent minimum river flow
	Mw	MGD	MGD	MGD	MGD
Lower Allegheny	1,150	750	25	12,500	2,000
Lower Monongahela	3,620	720	17	7,900	1,150
Ohio-Pittsburgh to Beaver R.	530	400	195	20,900	3,160
Ohio-Beaver R. to Pa. border	2,590	150	9	23,350	3,715
Total	7,890	2,020	246		

OCCUPATIONAL HEALTH ASPECTS OF FOSSIL-FUEL ELECTRIC POWER PLANTS

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OCCUPATIONAL HEALTH ASPECTS OF FOSSIL-FUEL ELECTRIC POWER PLANTS

Analyses of occupational health risks in fossil-fueled power plants have concentrated on raw material extraction (e.g. underground coal mining), transportation of the raw materials, and the health consequences of the air pollution from electricity generation. There is little information concerning occupational safety and health risks of the power plant workers.

The Edison Electric Institute estimates there are 1,000 fossil-fueled power plants in the United States, and that they employ approximately a half-million workers. A typical fossil-fueled power plant will have three to four units, each capable of generating fifty to one-hundred Megawatts. Most power plants are now using coal or are in the process of converting from oil and natural gas to coal for economic reasons and encouragement by the U.S. Department of Energy. This will result in greater occupational and environmental health risk because coal-generated power entails greater risk than oil or natural gas.

For a 1,000 Megawatt power plant, coal has a two to three times greater health risk (occupationally-related deaths) than an oil-fired plant, and a thirty-six to 1,120 times greater health risk than natural gas.[1]

From 1967 to 1971 at two 1,000 Megawatt TVA power plants (Colbert and Gallatin) there were 665,000 man-hours worked annually at each plant with 388.5 days lost because of accidents.[2] There were no fatalities. By contrast, in 1969 in coal mining there were thirty-two disabling injuries per million man-hours worked versus eight for all industries. There were 2,000 days lost per million man-hours worked which compares to approximately 258 for power plants.

Over half the health costs associated with the generation of electrical energy comes from coal mining; these risks will be only about 10 to 15% with strip mining. Coal transport by railroad may also represent hazards, particularly accidents, since 10% of all railroad cars are hauling coal to power plants (approximately five million carloads of coal in 1970).[2]

In comparing estimates of the health effects for alternative fuel cycles for equivalent 1,000 Megawatt electric units (Table 1), coal greatly exceeds oil and natural gas for occupational and nonoccupational deaths and occupational impairments.[1,3] The occupational death and impairment risks from coal are magnified (Table 2) when one considers the greater number of millions of Kilo-watt hours are generated by coal-fired plants compared to oil and natural gas in 1975. (844×10^6 for coal vs. 292×10^6 and 297×10^6 for oil and gas respectively). In evaluating the coal fuel cycle (Table 3), coal mining accidents and disease were the highest with power generation accidents very low, and power generation disease not even listed.

The future portends dramatic increases in coal production to meet energy needs, both in electricity and liquid/gaseous fuels. Coal mining will undergo major expansion in the Rocky Mountain states (Table 4) with a doubling of the number of existing mines over the next ten years with a four-fold increase in production.[4] Major power plants are planned (e.g. Intermountain Power Project in Utah) to produce power primarily for transhipment, or schemes (railroad, slurry pipeline) to ship the raw products to the Far West or Midwest. Considerable health risk and environmental degradation will likely occur from these energy needs - all of which will not be accomplished without considerable controversy.

The National Occupational Hazard Survey has provided a modicum of information on power station health hazards.[5] The National Occupational Hazard Survey was a two-year field study initiated by NIOSH in 1972, intended to describe the health and safety conditions in the American work environment, and to determine the extent of worker exposure to chemical and physical agents. The sample of businesses in the survey was selected by the Bureau of Labor Statistics and consisted of approximately 5,000 establishments in sixty-seven metropolitan areas throughout the U.S.

Hazard exposure data was provided for 453 occupational groups, including #433 - Electric power linemen and cablemen (43,433 workers) and #525 - Power station operators (3,340 workers). Table 5 illustrates the number and per cent of workers exposed to the more prevalent five individual hazards from the survey (continuous noise, mineral oil, ethanol, isopropanol, and trichlorethylene). Eighty percent of power station operators were exposed to continuous noise, and 23.5% were exposed to trichlorethylene.

Using the "Occupational Mortality in Washington State 1950-1971" study by Dr. Samuel Milham (NIOSH contract) as the reference, public utility workers have increased proportional mortality ratios (PMR) of cancer of the rectum (191), asthma (184), coronary heart disease (112), and accidents caused by electrical current (449).[6] (Table 6) Possible etiologies for such elevated PMR's other than chance or bias could be social class, sulfur-dioxide - particulate in-plant air pollution, electromagnetic radiation, solvent exposure, etc. An increased pulmonary embolism PMR in linemen may be secondary to traumatic injury.

In understanding the occupational health and safety problems of fossil-fueled power plants, two tours were arranged through electric generating stations. One was a public utility that burned coal, oil and/or natural gas, and the other was an industrial utility burning coal and natural gas. These tours provided practical experience in viewing the processes and work situations of power plants. Generally, the work environment did not appear particularly hazardous, although the noise level appeared excessive at numerous sites, and several of the maintenance jobs may be especially hazardous. One power plant was twenty-five years old with three units generating over 240,000 kilowatts, and the other was slightly older generating 175,000 kilowatts. Both employed over 100 workers with one-third to one-half working on maintenance.

Coal arrived in railroad cars from nearby coal fields - one unit consumed 1700 tons of coal per day. Inside a separate building, coal is shaken out of the railroad cars exposing the operator to coal dust and noise levels as high as 115 decibels. Ear protection was mandatory, and the building had been insulated with an acoustical material. No dust respirators were worn. Coal is transported by conveyor to a storage area; it is loaded and transported from here to a secondary storage area where coal is stockpiled (Figure 1). The conveyor belts may create a coal dust problem, particularly near adjacent walkways. The coal pulverizer is enclosed and utilizes large steel balls or a hammer mill. The powdered coal is blown into the boiler. Compared to natural gas, coal dust causes greater wearing of the pipes carrying steam in the boiler and results in greater maintenance problems (Figure 2).

In the boiler, air and coal are mixed to heat distilled water into superheated, pressurized steam (1005°F, 1,525 pounds pressure). Heat may be a problem, expecially if the boilers are inside a larger building. The steam is then transported to the main engine room to drive the turbines and generators to produce electricity. The turbine room was very noisy (estimated 85 decibels) (Figure 3). Nearby is the control monitoring headquarters. Older switches may contain mercury and present a mercury hazard to control monitor workers.

Flyash from the boilers may be a problem. It is considered a nuisance dust, and has a fine, sand-like quality with a variable silica content depending on the coal (17% at one of the power plants visited). Flyash exposure may occur at the base of the boiler when various doors are opened; a slight negative pressure within the boiler reduces dust exposures to flyash. A positive pressure would increase the dust exposure hazard. The flyash is conveyed to a separate building where it is moistened and hauled away via truck by contractors (Figure 4). Loading flyash may lead to excessive dust exposures, especially for farmers removing it to place on their fields to melt snow and hasten the planting season. Farmers prefer to load it dry to facilitate unloading. Electrostatic precipitators are used to remove particulate from flue gases going up the stack. Neither power plant had scrubbers to remove sulfur dioxide since they burned low sulfur ($<0.5\%$) coal. A comparison of stacks from two units of one power plant illustrates the differences in smoke from natural gas (left) and coal (right) fuels (Figure 5).

Power plants have two closed cycles for water use and re-use - condensors are used to condense the steam returning from the turbines. The water in the steam cycle is distilled and kept free of impurities to reduce scale formation. Cooling water from cooling towers passes through the condensor; this water is also purified - a large water treatment plant exists which may entail exposures to chlorine gas, sulfuric acid, and sodium hydroxide. Settling ponds are used prior to any water discharge. A wet chemistry laboratory is used to monitor water purity.

Maintenance work may prove particularly hazardous since it is an ongoing activity throughout the plant. Exposures are usually intermittent, but may be quite heavy. One of the generators was being overhauled on one tour; solvent and oil exposures may occur here (Figure 6). Trichlorethylene and methyl ethyl ketone exposures are not unusual. Insulation on pipes may contain asbestos; this is more common in power plants built in the pre-1970 era (Figure 7). Removal of the asbestos may generate a hazardous dust exposure. Scale and plaque that accumulates on steel parts may be sandblasted resulting in a silica hazard. A welding shop on the premises has exposures to various welding fumes.

The electricity generated passes through transformers prior to distribution. The transformers may contain polychlorinated biphenyls, and an exposure may potentially occur during maintenance. The electricity then passes through a sub-station where electromagnetic radiation exposures may occur.

A list of the safety mishaps at one power plant was available for February - it provided a typical listing of power plant injuries: An apprentice mechanic received a hairline fracture to his right arm when the sump pump he was removing slipped and fell on him. A test and instrument journeyman received medical attention for inhalation of chlorine gas. A serviceman received flash burns to both eyes when a flash occurred as he was installing jumpers in a meter base. A skilled helper received a laceration to the two middle fingers on his left hand when he caught them between the forks on a forklift. Five meter readers received medical attention for dog bites.

Measurements of crystalline silica, respirable coal dust, total coal dust, and fly ash were made at three coal-fired power plants in Colorado by NIOSH. Only a small number of workers were in direct contact with coal or flyash. Personal samplers were used; coal handlers were exposed to excessive total coal dust at all three power plants and to excessive silica at two. (Tables 7 and 8). One of the power plants required NIOSH-approved respirators for its coal handlers. Most coal handlers worked outside on a high stockpile of coal where engineering controls were not feasible. Excessive flyash exposures occurred at one utility plant among utility men cleaning baghouses (Table 9).

Professor N. LeRoy Lapp has reported pathological findings on a lung biopsy of a forty-two year old boiler repairman who worked at a power generating station that burned bituminous coal.[8] He was a nonsmoker who had been employed for twenty-four years. His chest x-ray (Figure 8) was read as normal. The lung biopsy was obtained at the time of mitral valve replacement. Light microscopy (Figure 9) revealed mild-moderate thickening of the walls of pulmonary arterioles, and focal areas of thickening and fibrosis of alveolar septae. There were also a moderate increase in the number of pigment-laden alveolar macrophages. Electron microscopy (Figure 10) revealed thickening of the basement membrane in the alveolar walls by fibrous material and collagen fibers. Four types of particulate matter were found in the interstitium (Figure 11): 1) dense needle-like material, 2) smooth, angular pieces, 3) larger rectangular chunks, and 4) granular aggregates of the previous three types of particle. Also, small, round debris (probably silica) and a few asbestos bodies within macrophages were noted. Presumably, this particulate material represented an occupational exposure to mixed dusts (flyash, asbestos, silica, coal dust) in a nonsmoking boiler repairman.

The Federal Power Commission estimated 3,607,000 tons of flyash were released to the atmosphere in 1972 from 696 major steam plants.[9] This resulted from the combustion of 350 million tons of coal with an average ash content of 13.4 percent (by weight). Recently, flyash has been reported to be mutagenic. Both organic and inorganic compounds were found in a respirable fraction of coal flyash that caused frameshift mutations in a bacterial strain lacking normal excision repair (positive Ames test).[10] A horse serum filtrate had approximately a tenfold greater activity than a saline filtrate of coal flyash suggesting this increased the sensitivity of the Ames technique in detecting mutagenicity of complex mixtures, or enhanced solubility of mutagenic compounds, or provided serum proteins for complexes to be formed increasing mutagen detection. Substances on the surface of flyash deposited deep in the lung could be similarly soluble in alveolar fluid. The surface of flyash may contain polynuclear aromatic hydrocarbons (shown to be mutagenic and may bind serum proteins), and may condense many metals, e.g. cadmium, selenium, arsenic, antimomy, molybdenum, lead, nickel, beryllium, manganese, and iron.

Coal-fired power plants may result in population doses of radioactive particles and daughters of uranium 238 and 235 and thorium 232 that exceed those of a comparably-sized nuclear plant.[11] These particles are primarily attached to flyash. Radium-226 and radium-224 are the major contributors to the whole-body and most organ doses from the coal-fired plant, and ingestion is the main exposure pathway.

Coal and oil-fired power plants are responsible for nearly two-thirds of the nation's sulfur dioxide emissions. Seventy-four percent of 623 large coal and oil-fired power plants are currently in compliance with sulfur dioxide limitations.[12] Sulfur dioxide is an irritant to the respiratory mucosa, primarily to the upper respiratory tract where it is absorbed as sulfurous acid or one of its ionization products. In high concentrations, it may cause a chemical pneumonitis. Frank and associates studied acute changes in pulmonary flow resistance after ten to thirty minute exposures in healthy human volunteers.[13] Pulmonary flow resistance increased significantly in 39% exposed to five ppm, 72% to thirteen ppm, and in only one of eleven exposed to an average level of one ppm. The change occurred within one minute of exposure, increased after five minutes, but on the average, showed no further increase after ten minutes. The cause of these changes appeared to be bronchoconstriction rather than tissue swelling or edema. Smith and Peters assessed the effects of chronic, low-dose sulfur dioxide exposure on pulmonary function.[14] In studying 113 copper smelter workers, they noted that at average levels of 1.0 and 2.5 ppm, sulfur dioxide was associated with an excess loss of FEV_1 over one year and an increase in respiratory symptoms after controlling for smoking. No exposure to respiratory particulates on pulmonary function was found. Workers with FEV_1 less than the predicted values based on age and height showed evidence of even greater losses of pulmonary function related to sulfur dioxide exposure.

Trichlorethylene exposure may occur among maintenance employees; the main physiological response is central nervous system depression, especially from acute exposure. Other effects include visual changes, confusion, euphoria, incoordination, nausea, skin irritation and alterations in hepatic and renal function. Liver cancers in mice fed trichlorethylene by gastric intubation have been reported by the National Cancer Institute.[15]

Exposures to polychlorinated biphenyls may occur to workers repairing electrical capicitors and transformers. About 95% of the 100 million capacitors produced annually in the U.S. contain PCB's, and the 135,000 PCB-containing transformers represent 5% of all transformers in the U.S.[16] The Toxic Substances Control Act of 1976 has now banned manufacture, processing, and distribution in commerce of PCB's. Health effects of PCB's were best described from the Japanese Yusho incident in 1968 where 1,200 cases were reported from persons exposed to up to 3,000 ppm of Kanechlor 400 in rice bran oil contaminated by PCB's during a heat exchanger leak.[16] Signs and symptoms included darkened skin, acneiform eruptions, including chloracne, eye discharges, nausea, visual changes, neurological changes, and edema and/or low birth weight among infants born from women exposed to PCB's during pregnancy. PCB's may induce hepatic microsomal enzymes, and in animals, may cause hepatic necrosis, hepatic tumors, and an altered immune response.[16]

Power plant workers may be exposed to electromagnetic radiation since there are sixty Herz electric and magnetic fields in several locations on the power plant premises. Electromagnetic radiation may cause biological effects from tissue heating, or by membrane excitation which occurs earlier. Studies of electromagnetic radiation effects have directed attention to growth and development of plants and animals, cellular and molecular aspects of cell metabolism and physiology, chromosomal changes, alterations in behavior, and the health of utility linemen.[17-18] The Johns Hopkins University nine-year study of utility linemen working on energized high voltage transmission lines found no physical, mental, or emotional changes attributable to the exposure.[19-20] Soviet studies of workers exposed to electromagnetic radiation in substations and switchyards found increased subjective findings, e.g. headache, fatigue, neurological and cardiovascular changes, hematological changes, and decreased sexual potency.[21-23] Marino and Becker have found that rats exposed to a sixty Herz electric field for one month exhibited hormonal and biochemical changes similar to those caused by stress.[24] In another experiment they continuously exposed three generations of rats to the same electric field and found increased infant mortality and stunted growth.[24]

A preliminary study in man showed that a 1-G (45 Herz) magnetic field has a slight but statistically significant effect on cognitive skill assessed by the Wilkinson Addition Test and Response Analysis tests.[25] Constant magnetic fields are reported to slow wound healing by a delay in fibrosis and fibroblast production.[26]

In summary, there are multiple potential and real hazardous exposures for fossil-fuel electric power plant workers. There is a paucity of published information on the prevalence of occupational disease and injury rates among these workers. The working population is generally stable, and would provide ample opportunity for research. An important study design feature would be to isolate the many potential exposures, in order to study workers exposed to single hazardous agents. This may be particularly difficult to achieve in maintenance workers. Further studies on the health status of power plant workers are needed.

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TABLE 1

COMPARISON OF HEALTH EFFECTS FOR ALTERNATIVE FUEL CYCLES*

Effects	Coal	Oil	Natural Gas	Nuclear
Occupational deaths	0.54-8.0	0.14-1.3	0.06-0.28	0.035-0.945
Nonoccupational deaths	1.62-306.0	1.0-100.0	...	0.01-0.16
Total deaths	2.16-314.0	1.1-101.0	0.06-0.28	0.045-1.1
Occupational impairments	26.0-156.0	12.0-94.0	4.0-24.0	4.0-13.0

*For electric power production per 1,000 megawatt electric units.

TABLE 2

COMPARISON OF HEALTH EFFECTS FOR ALTERNATIVE FUEL CYCLES*

Fuel	1975 kW-hr, Electric Units X 10^6	Equivalent No. of 1,000-Megawatt Electric Plants	Estimated Deaths		Estimated Occupational Impairments
			Occupational	Nonoccupational	
Coal	844	128	69.0-1,024	207.0-39,168	3,330-20,000
Oil	292	44	6.0-57	44.0-4,400	530-4,100
Gas	297	45	3.0-13	...	180-1,080
Nuclear	168	26	0.9-25	0.3-4	100-340
Total**	1,601	243	79-1,119	251-43,572	4,140-25,000

*For electric power production in the United States in 1975.
**Some totals do not add up because of rounding.

TABLE 3

ESTIMATES OF HEALTH EFFECTS OF COAL FUEL CYCLES

Procedure	Occupational Deaths*	Occupational Injuries and Disease*	Nonoccupational Deaths
Coal fuel Extraction Accidents	0.45-1.24	22.0-80.0	...
Disease	0.00-4.8	0.6-48.0	...
Transport Accidents	0.055-1.9	0.33-23.0	0.55-1.3
Processing Accidents	0.02-0.05	2.6-3.1	1.0-10.0
Power Generation Accidents	0.01-0.03	0.9-1.5	...
Air Pollution	...	...	0.067-295.0
TOTAL	0.54-8.0	26.0-156.0	1.62-306.0

*Per 1,000 megawatt electric units per year.
Some totals do not add up because of rounding.

Table 4 The magnitude of the expansion of coal mining in far Western states.

State	Number of Existing Mines*		Number of Planned New Mines	Annual Coal Production**	Increases in Production**
Arizona	2	(0)	0	10.2	5.0
Colorado	33	(18)	37	9.4	45.6
Montana	8	(0)	6	26.1	48.2
New Mexico	5	(1)	3	9.8	77.7
North Dakota	10	(0)	5	11.1	42.6
Utah	20	(20)	25	7.9	64.5
Washington	4	(1)	2	4.1	2.0
Wyoming	20	(5)	23	30.9	139.8
Total, 8 states	102	(45)	101	109.5	425.4

* Numbers in parentheses indicate underground mines; remainder are strip mines.

** Annual production in millions of short tons per year. A short ton, the industry standard measure, is very nearly equal to a metric ton, or 1000 kg. Data are current for 1976.

TABLE 5 NATIONAL OCCUPATIONAL HAZARD SURVEY

		#	% of the occupational group
	1) Continuous Noise		
433	Electric power linemen and cablemen	2,881	6.6%
525	Power station operators	2,673	80.0%
	2) Mineral Oil		
433		26,988	62.1%
525		1,159	34.7%
	3) Ethanol		
433		1,984	4.6%
525		209	6.3%
	4) Isopropanol		
433		3,229	7.4%
525		488	14.6%
	5) Trichlorethylene		
433		11,595	26.7%
525		789	23.6%

TABLE 6

CAUSES OF DEATH OF ELECTRIC UTILITY WORKERS

Cause of Death	Proportional Mortality Ratio
Public Utility Workers	
Cancer of the rectum	191
Asthma	184
Coronary heart disease	112
Accidents caused by electrical current	449
Linemen	
Cancer of the large intestine and the rectum	157
Cancer of the respiratory system	129
Pulmonary embolism	278
Accidents due to falls	239
Accidents caused by electrical current	2,657

TABLE 7

BREATHING ZONE AIR CONCENTRATIONS OF TOTAL COAL DUST

NIOSH Health Hazard Evaluation

Report

Sample Number	Location	Job Classification	Time of Sample	Total Coal Dust (mg/M_3)
61	Basement	Equipment Operator	8:12 am - 2:30 pm	0.7
115	"	Bag House Cleaner	8:28 am - 3:02 pm	0.5
111	"	Utility	8:17 am - 9:20 pm	56.0
64	"	Heavy Euqipment Operator	8:20 am - 2:25 pm	64.0
117	"	Utility	8:30 am - 12:00 noon	39.0
60	"	"	12:30 pm - 2:30 pm	1.6
			EVALUATION CRITERIA	10.0 1/

1/ = the nuisance dust TLV of 10 mg/M_3 was used, since these were not respirable samples

TABLE 8 BREATHING ZONE AIR CONCENTRATIONS OF RESPIRABLE CRYSTALLINE SILICA (QUARTZ & CRISTOBALITE) AND RESPIRABLE COAL DUST

NIOSH Health Hazard Evaluation Report

Sample Number	Location	Job Classification	Time of Sample	CRYSTALLINE SILICA		
				Quartz (mg/M_3)	Cristobalite (mg/M_3)	Coal Dust (mg/M_3)
2381	Coal Handling	Coal Handler	8:07 am - 3:00 pm	0.14	*	3.0
			EVALUATION CRITERIA	0.05	0.05	2.0
			LABORATORY LIMIT OF DETECTION	0.03mg/sample	0.03mg/sample	---

TABLE 9 BREATHING ZONE AIR CONCENTRATIONS OF FLY ASH

NIOSH Health Hazard Evaluation Report

Sample Number	Location	Job Classification	Time of Sample	Fly Ash (mg/M_3)
110	Outside Coal Handling	Bag House Cleaner	8:20 am - 2:25 pm	1.7
59	" " "	Welder (Cleaning Bag)	8:17 am - 9:20 am	83.0
			EVALUATION CRITERIA	10.0

Figure 1 Coal conveyer, loading, and storage processes

Figure 2 Coal-fired boiler

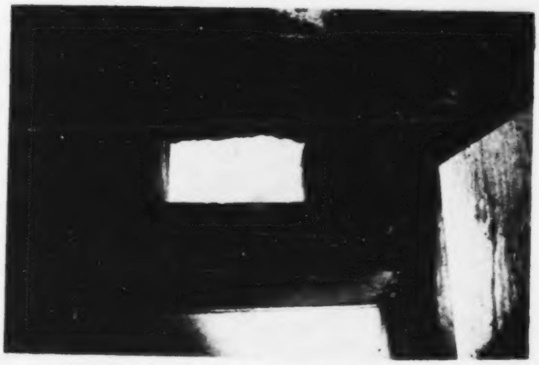


Figure 3 Turbine and generator room of a fossil-fuel power plant

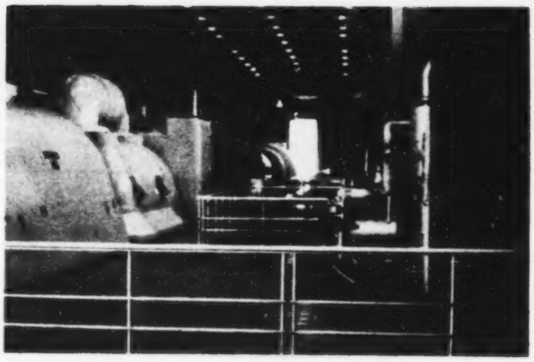
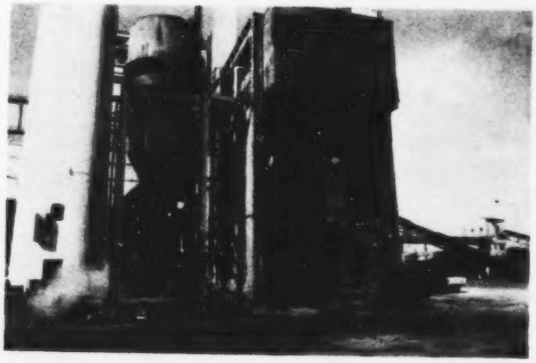


Figure 4 Flyash disposal loading facility



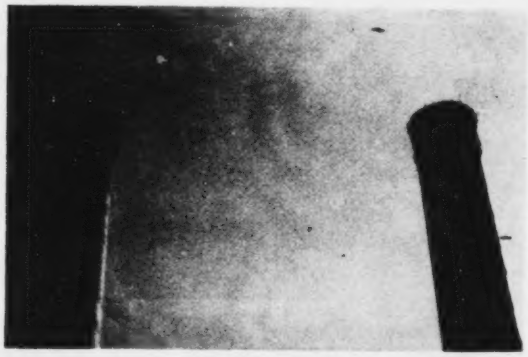


Figure 5 Smoke from a stack from a boiler burning natural gas (left) compared to smoke from a stack from a boiler burning coal (right)

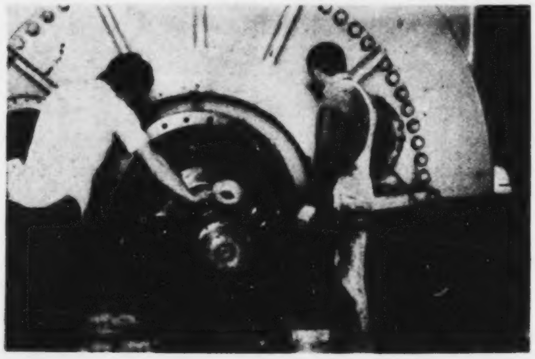


Figure 6 Maintenance workers overhauling a turbine

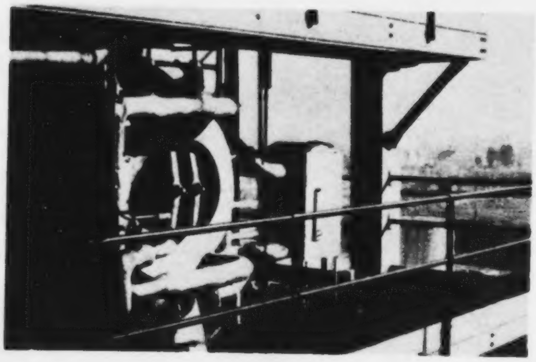


Figure 7 Older pipe insulation may contain asbestos posing a hazard during replacement

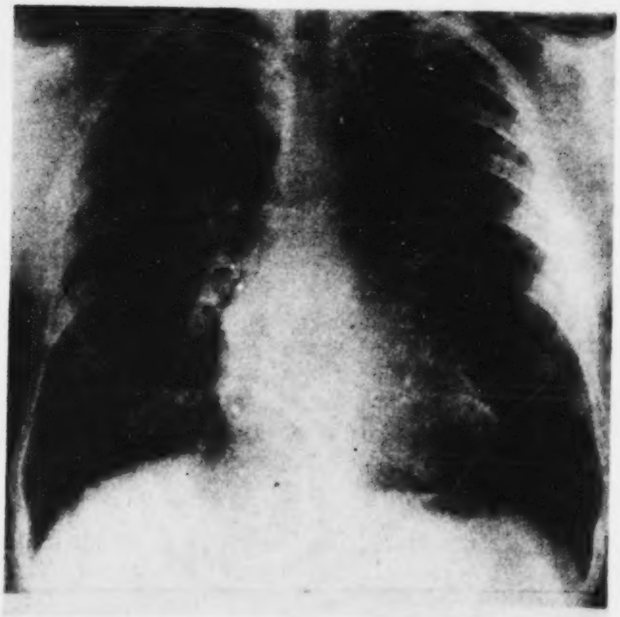


Figure 8 PA chest radiograph of 42 year old nonsmoking boiler repairman (read as 0/1 on the ILO/UC 1971 classification of the radiographs of the pneumoconioses)

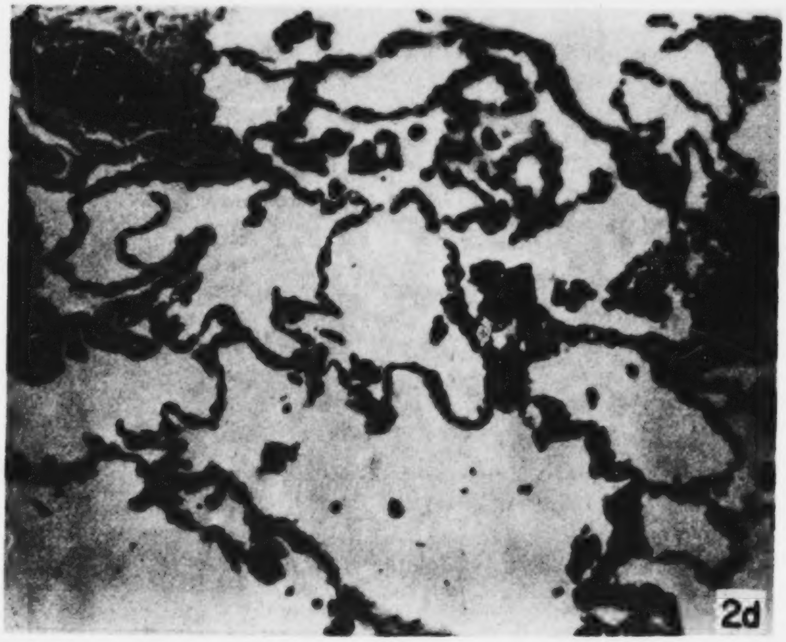


Figure 9 Lung biopsy specimen demonstrating mild-moderate thickening of the walls of pulmonary arterioles, and focal areas of thickening and fibrosis of alveolar septae

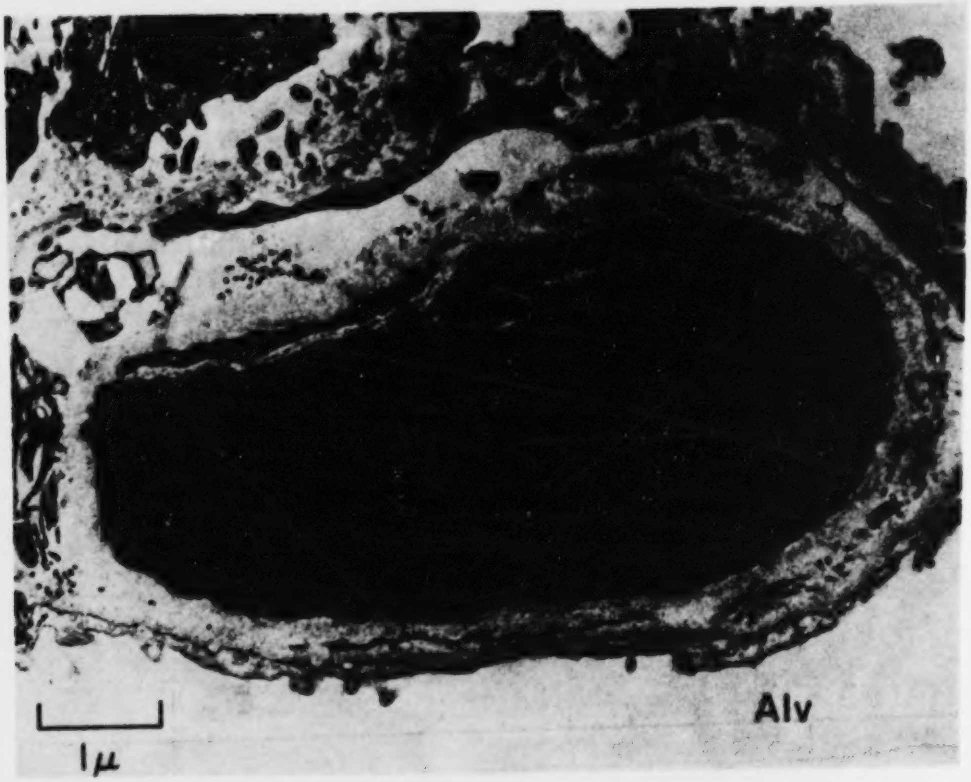


Figure 10 Thickening of alveolar septae due to collagen fibers and fibrous material (EM x 20,000)

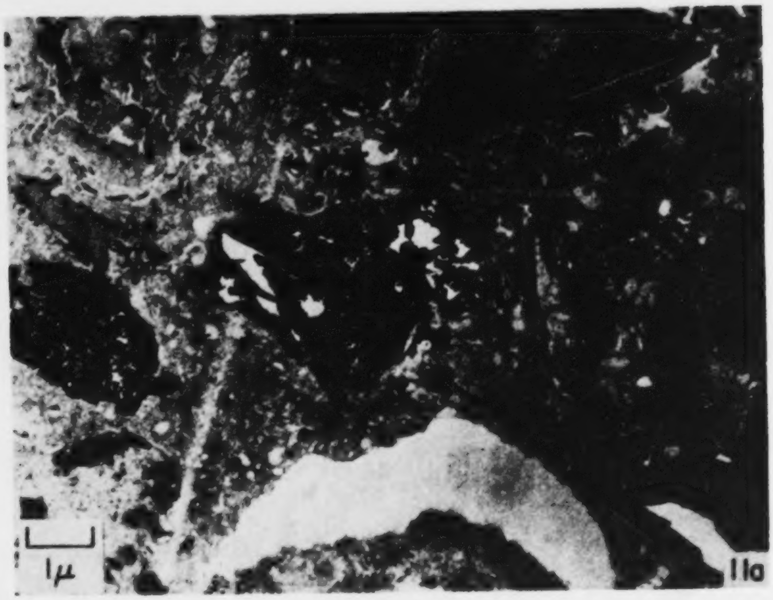


Figure 11 Particulate matter found in the pulmonary interstitium: N-needle-like material, An-angular pieces, R-rectangular chunks, and Ag-granular aggregates (x 9,400)

DISCUSSION

SESSION III

Dr. L. Hamilton: I would like to make a general clarifying comment particularly in view of the remarks that have just been read into the record. Particularly in the light of Dr. B. Ferris's presentation because I think that some confusion could arise. You know there is no evidence that SO2 does anybody any harm. Of course, we accepted that in the light of present day epidemiology that indeed harm is from the particle material. But SO2 is mainly oxidized to another material, sulfate or sulfuric acid; and it is these materials that are harmful. But SO2 is the harmful precursor; it isn't the material itself. In essence, I think it would be very hazardous to give the public the impression that we are seeing no effect of SO2, and that we don't have to worry about the material itself--it is what SO2 turns into. Now there is another very important consideration that we have to keep before the public and that is when SO2 leaves the stack as a gas, in the process of being oxidized to sulfate, it forms a respirable particle. This is another important matter. The very best particulate removers in the world will not remove the SO2 and prevent the occurrence of this respirable particle in the atmosphere. And a third important point is that, if it is a sulfate particle, it's capable of being carried over many thousands of miles. To give you an example, it has been shown that in Europe, long-range transport of the acid material has been amply demonstrated by European workers. Now, this is a very important problem; the fact that you have long-range transport. It means that if you have a 50% reduction in SO2 emissions in New York, you can do the sort of study that has just been reported, in which the SO2 levels apparently have gone down and one hasn't seen any concomitant change in health. It is easy again by just focusing sometimes on isolated studies, to draw misleading conclusions. I mention these things in a very simplistic way, at this time, because I would like the ORBES study, which is going to be the subject of consideration, to take these into account. I feel it is necessary that these considerations be clearly enunciated at this time. But, of course, tomorrow I would like the opportunity of dealing with these uncertainties more fully.

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Dr. H. Spencer: I think in all fairness I should respond to and compliment Dr. Ferris and his analysis of the work of Lave and Seskin in discussing the regression correlation techniques used. I respond by saying that the classic misstatements and mis-use in that technology exist in engineering literature in the classic textbook by Metcalf and Eddy on Water and Wastewater Treatment, and the text by Nemerow on Industrial Wastewater Treatment. I think that they take a stand on the issue mentioned, that certain regressions did not show any relationships, and all of these regression techniques take a partial of the sum of the squares which, in turn, makes the assumption that the variables therein hold constant relation to each other, which is totally wrong. I have a question I'd like to address to the group. That is in relation to, not knowing what sulfur dioxide does except that in sulfuric acid there is another gaseous component which was passed by rather lightly this morning, the subject of nitrous oxides--better known as NOx. Among that family of compounds it is quite reasonable to write equations about the formation of nitrous acid which, in times past, in the good years of molecular biology, since it was written as HONO was known as "Oh NO." Nitrous acid is a powerful mutagen. That is a known fact. It is a fact in molecular biology and has been for many years. We need not argue that. I would like to hear an answer to the question: what is the effect of nitrous acid, through inhalation into the alveoli of the lung?

Dr. Ferris: I don't know that there have been any measurements of nitrous acid *per se*. By and large, NO has been felt not to have any health effects. It is oxidized to NO_2, and that in turn is hydrated to H_2NO_3 so it's usually the nitric acid that is of concern.

Dr. H. Spencer: The problem I have with that is the long term latency of cancer and the fact that we cannot show an effect by respiratory volume or irritation of anaphylactic shock, of which the person may be suffering. To me it is a meaningless measure of the possible impact of that one particular thing.

Dr. Ferris: But if you are talking about the carcinogens, we are faced with all sorts of competing factors and, with the long latent period, it is awfully hard to look back in your "Retrospectroscope" to determine which is the initiating agent, agents, or co-agents.

Dr. H. Spencer: I can't argue with that.

John Blair, Evansville, Indiana: One of the things I'd like to hear this panel address, both on air and water pollution, is the synergistic effects of all these chemicals forming together, and what effect do these have on health problems of people? Also one other question about power plants, loss of fuel in power plants and its relation to ozone productions. That seems to be one of the major elements and the EPA saw fit to change the standards.

Dr. Andelman: I hesitate to really comment very much about the synergistic health effects. That's certainly not my area. I have been somewhat familiar with polycyclic aromatic hydrocarbons and their possible influence on waterborne disease, and have been looking at some of the health effects that other people measure. Of course, some of these chemicals are potent carcinogens. There have been a number of studies which not only have attempted to determine their carcinogenicity on an individual basis, but on some of the mixtures of polycyclic hydrocarbons as well. In some such studies that have been recently reviewed, it has been shown for some of the potent carcinogens like benzopyrene and dibenzanthracene that, if indeed you put these in with mixtures of other polycylic aromatic hydrdocarbons, there doesn't seem to be either potentiation or cocarcinogenicity.

Dr. Rom: I'm not aware of any measurements of ozone on the premises of power plants. Most of the ambient ozone measurements are made in communities. It generally has been ascribed that most of this ozone has been due to various transportation systems and reactivity from sunlight. Ozone may have acute respiratory effects. Most of these effects have been demonstrated by the investigators of the Los Angeles Basin. There have been several recent studies that are probably worth commenting on and would certainly warrant further discussion. One is whether there is acclimatization to ozone, whether people who live in high ozone environments for an extended period of time have less of an acute response to experimental ozone exposures than those who live in areas with less ozone. I would think that is a recent finding that would warrant further discussion.

Dr. Ferris: I would like to amplify on what Dr. Rom said. The NO-NO2 interaction in the presence of ultraviolet can release atomic oxygen which then reacts with molecular oxygen to form the ozone. This is part of the photochemnical reaction and requires sunlight (ultraviolet) to cause the interaction. The effect of tolerance to ozone has been demonstrated in chamber studies that have compared Los Angelenos, who had lived there for some time, with Canadians, who came to Los Angeles for the study. The Canadians showed much more reaction to the chamber exposure than the Los Angelinos who had been exposed to it over a period of time. This tolerance has been used as an explanation as to why we have not seen much of any effect of ozone increases during alerts in the Los Angeles area. To speak more specifically to possible interactions, this of course, is of great concern because frequently in the laboratory you give gas A; then you give compound B; you don't usually give them as a mixture. It is only just recently that some of the chamber studies have used various mixtures. We also have to refer to a certain number of animal studies to assess this and to identify synergism or antagonism. Dr. Amdur in Boston, Dr. M. Corn here in Pittsburgh, and others have looked at the interaction between SO2 and particulates. They have shown that with hygroscopic type particulates and certain chemicals, such as metals that can act as a catalyst, they can augment the effect of the SO2. Presumably the SO2 is absorbed on the particle, and then with the catalyst and water vapor it is converted to sulfuric acid, which is much more toxic than the SO2. Related studies have looked at the interaction of SO2 with ozone and SO2 with hydrogen peroxide and in both instances presumably the end product has been a sulfuric acid mist which again apparently is more toxic than the original SO2 gas. I certainly underscore what Dr. Hamilton said that the SO2 is a precursor and probably its end product in the final common pathway is its appearance as a sulfate that we have to be concerned about. But do not fall into the next trap which says that all sulfates are equally bad, because they are not. I think that is why we should not set a sulfate standard, until we can improve our chemical techniques so we can specify the type of sulfate we are talking about.

Dr. Stukel: There are documented cases of elevated oxidant levels due to the long range transport of power plant emissions. In a recent study, the pollutant concentrations in a St. Louis area power plant plume were monitored, over a long distance, using plume tracking aircraft. It was found that the oxidant levels in the plume increased over time, and that a large fraction of the elevated oxidated levels measured in Springfield, Illinois, could be attributed to this plume. Springfield is more than a hundred miles north of St. Louis.

Dr. Ferris: Is this not due to interactions of oxides of nitrogen in the plume?

Dr. Stukel: Yes, that's correct. The point is that oxidant levels are usually thought to be only due to automobile emissions. I wanted to make the observation that this is not always the case.

Dr. S. Ali, Public Service of Indiana: I have a question for Dr. Rom. The material that was shown was on smaller units which were 25 years or older. I wonder if he intended to show the worst case situation rather than the actual situation which exists in most power plants constructed and in operation?

Dr. Rom: Of the two power plants that I toured, the worst case was the industrial one for which I didn't have any slides illustrating the problems there. They would not allow photographs. The other one, a public utility, had two of its units that were very old; the newest was the largest of the three and was constructed during the early seventies. The other two plants were built in the 1951-1955 era. It reflects the technology that existed 20-25 years ago. And in that sense, I would say it is more of a worst case example than a best case example. There are quite a few large coal-fired power plants being built in our region, and I'm told that many of the problems that I illustrated no longer exist. For example, they don't use asbestos-containing insulation on the pipes; that's being taken over by fiberglass. Also I was told that they have electric switches that do not use mercury so that there is no mercury hazard. I am not sure about the sandblasting operations. They are, however, having a lot of coal dust problems, and I'm told that their primary concern is now the dust hazard in the new, large power plants. I think what may happen in many of these cases is that as we advance with our newer technologies, we have new problems to solve.

Dr. Ali: Another question I have for you, Dr. Rom, is that you're trying to show here that smaller units are prone to more problems. This is somewhat contrary to the objective of EPA "Prevention of Significant Deterioration" which encourages going to smaller noncentralized units. Do you have any views on the subject? And have you provided your views to EPA?

Dr. Rom: The answer to the second question is no. To the first part of your question--True, smaller plants may have more problems. I think that is more in terms of how new a plant is and newer plants tend to be larger plants. Many of the older problems are solved with newer technologies.

Dr. Ali: The trend would be to smaller units if "Prevention of Significant Deterioration," especially in the midwest and east, becomes the main consideration.

Dr. Radford: I have a couple of comments and then a question for each of the panelists. With regard to Dr. H. Spencer's question about nitrite arising from oxides of nitrogen when the nitric acid or NO2 hydrolizes, it is my understanding you get both nitrite and nitrate molecules, which might suggest that nitrite could be significant. We have some unpublished data on smokers, who inhale a lot of oxides of nitrogen too, along with all the other stuff. One would predict that if nitrite were a significant product in a smoker, you would get a very definite relationship between the production of the methemoglobin, which is generated by nitrite in the body, and the amount of exposure to other agents such as carbon monoxide.

We found very slight effects in a very large sample. We are currently working on people taken from all over the United States. We are finding a very slight effect of levels of smoking on the production of methemoglobin in the body. It has been a much argued point. But, it is extremely small and it suggests to me that however the chemistry goes, the nitrite component must be extremely small. This fits with what Dr. Ferris said about NO also being present because we find no physical logical evidence. That's the comment I wanted to make. I'd just like to make a quick question. Dr. Ferris. Would you like to say anything about the Six City Study you're currently doing? Would you want to make any comments or preliminary statements, or have you said enough?

Next question is to Dr. Andelman: Is there any extensive use of river water for irrigation purposes where some of these contaminants in the river, before treatment in water systems, might get on to food crops and therefore have a pathway into the human population? Finally to Dr. Rom, and I guess it's really relevant to the point made by a previous discussant about the age of power plants. There was a technology--it may no longer exist--where in coal-fired power plants, the feeding of the fuel into the boiler was under positive pressure. I have been in one such plant, and those are extremely dusty because we have the fuel fed at higher pressure at any place in the working area. Does anybody have any idea how frequently this positive pressure technology is being used?

Dr. Ferris: Reluctantly I will make some preliminary comments recognizing that we are still finding and collecting data much faster than we can analyze it. One of the things we are finding which is extraordinarily important, which others have commented on, is that the levels that to which we are actually exposed are much more influenced by the indoor environment. If you want to predict what a person's exposure is going to be, you have to measure the indoor environment, which is much more likely way to give an accurate exposure estimate than the outdoor environment. Yet the outdoor environment is what we are controlling. Also, when we selected our cities, we had a nice gradation of exposures to total suspended particulates, from very low to quite high which is one of your neighboring communities, Steubenville, Ohio. We started collecting mass respirable particulates and find that this nice separation no longer exists. At this time, the cities are very similar in mass respirable particulates and Steubenville, Ohio, sticks out as the only one high in mass respirable particulates. So this is another factor that needs to be taken into account. Although there may be a lot of big stuff in the total suspended particulates, they may be having no effect on the health. We have finished analyzing some data from Steubenville, Ohio, which involves hospital admissions over four months per year, over a four year period, and tried to link this to levels of pollution on the alert days. There were days when the pollution levels ran over 300 to 400 micrograms, total suspended particulates and SO2. We were not able to demonstrate any association between levels of pollution and non-traumatic hospital admissions. We also looked at oxides of nitrogen and some spotty ozone data. We were not able to see that they were having any influence on hospital admissions. This study can be criticized that perhaps the population base is too small to show anything, or that we are using too coarse an endpoint for this since all we could look at were the total nontraumatic hospital admissions. The other intriguing thing that has come out is that if homes are using gas stoves for cooking, as other people have shown, there are higher levels of oxides of nitrogens throughout the home. This is so even though the stove is being used only three or four hours in the course of the day. It affects the 24-hour level. Other people have looked at the instantaneous values and they have been as high as almost 1 part per million over 15 minute periods of time. It may very well be that these peaks are much more important because the levels that we see in the homes are way below the 24 hour annual standard for NO2. There are some suggestive data that this may be having some effect upon the respiratory symptoms and tests of pulmonary function in the children who live in those homes. These are very, very preliminary and we need to have further evaluation of it. We are not seeing marked differences in respiratory symptoms or pulmonary function in the adults across the six cities. I think we need to look at these on a longitudinal basis so we don't have the high inter-city variability to mask intra-city effects. The analyses are still in a very preliminary stage.

Dr. Andelman: I have no factual information about the possible use or impact of the use of wastewater from power plants or cooling water in terms of their ultimately being used as irrigation water. There are a couple of people at the Graduate School of Public Health attempting to develop information about the possible impact of power plant effluents on health. I wonder if any of them know of papers reviewing the subject since they have been reviewing this literature much more extensively than I? Do any of you have any ideas about this?

J. Bern, Student, University of Pittsburgh: With regard to irrigation use of power plant effluents, the many studies done by EPA, Corps of Engineers and DOE were concerned with the release of heavy metals from the ash, as well as from FGD-scrubber sludges. There has been some concern with the uptake of cadmium and vanadium by plants. The researchers were concerned with plant health rather than with the problem of heavy metals entering the food chain. With regard to sewage effluents, there are limitations placed on the spraying of sewage effluents or digested sludge or untreated wastewaters on food crops. The limiting factor used is cadmium concentration. This limitation is due to the expected uptake by the plants as a result of a specific concentration of cadmium. There are a number of studies with a great deal of information with regard to heavy metal uptake. Now, as far as organics are concerned, I believe that very little information is available in literature or in the reports of actual operations.

Dr. Rom: The power plant I toured had negative pressure rather than positive pressure for its coal dust. And I don't know how prevalent positive pressure is throughout the country. Perhaps the utility industry has information on that. At a number of coal fired power plants in the west, I'm aware of some irrigation experimentation that has occurred with wastewater. This has been done in Utah by Utah State University. They have been concerned more about the effects on the plant rather than toxic products entering the food chain. Their primary concern has been with vanadium. However, toxic effects have been observed.

Dr. Stukel: Are there any comments on positive pressure technology that any of you would like to make?

Dan Swartzman, University of Illinois School of Public Health: Professor Shapiro mentioned that you were hoping to have a document to represent the state of knowledge at this time, and so I would like to offer some results that were obtained by a colleague of mine at the University of Illinois, School of Public Health, with two very important caveats. One is that at this time the report which has been done under contract with the United States Environmental Protection Agency has not been approved and released by EPA, although a public presentation of the data was made. Secondly, this is based on my understanding of that public presentation and if you are willing to accept my understanding of it, then I don't want to trust your judgment too far. I offer this not as expert testimony but just pointing out some information that does exist. And what ought to happen is that somebody ought to look into it if there is any interest after my presentation.

Professor Tsukasa Namikata was asked to look at a 14% reduction in the mortality rate that occurred in the Chicago area between 1971 and 1975. Specifically, he was asked whether or not he could associate any of that reduction with reduction in air pollution levels in the city. He divided the city into 76 recognized community areas. He then took results from 28 particulate monitors and 24 sulfur dioxide monitors and extrapolated air pollution results for the community areas that did not, in fact, contain monitors. He noted in the presentation that that was probably not too bad for particulates. That sort of extrapolation got a little bit tenuous when you dealt with SO2. He then did a similar linear regression technique to what was done by Lave and Seskin, and controlled for socioeconomic variables, educational levels and an index of other environmental stresses. He had three indexes which he gave the sources for, and I'm afraid I do not have them at this time, what the indexes were or where he got them. He looked at the total suspended particulates, sulfur dioxide, a combination of total suspended particulates and sulfur dioxide, and a complex of total suspended particulates and sulfur dioxide. So, there are four different categories of pollution. He also looked at a wide variety of causes of death, including just about everything I could think of as a non-doctor other than accidents; he excluded accidental deaths and homicides. The findings on mortality rates of the study were that 5% of the 14% drop in mortality rate from 1971 to 1975 was associated, at a statistically significant level, with a 25% reduction in total suspended particulate levels. I don't know about the morbidity aspects of the study--he didn't present them at the presentation I attended--but I understand in discussing this with some of the people who worked with him, that they did find some significant associations with reduction in pollution levels and hospital admissions. I just offered that for the sake of the record and those interested should contact Dr. Tsukasa Namikata. Again, I'm not sure whether the study has been released yet. In fact, when EPA reviews it, they may decide they should not release the study.

Dr. S. Morris, Brookhaven National Laboratory: I have two questions. One for Dr. Rom: you start by pointing out that in the estimates of health effects throughout the total fuel cycle, there were no estimates that had been previously made for occupational disease in power plants. Now that you have gone through this, do you have any preliminary estimate of the incidence of occupational disease--say, per power plant per year, or something--that might be put in there. The second question is to Dr. Ferris: in your discussion of the statistical reanalysis of the Lave and Seskin data, I understood that in general the result was to confirm the association that Lave and Seskin found, but then you followed by saying that your conclusion was there was not any likelihood there of an effect at current levels of air pollution. What I'd like to ask is, is that conclusion based on your thinking that the association of Lave and Seskin is not causal, or because of the nonlinear damage functions you mentioned, or for some other reason?

Dr. Rom: I don't have any preliminary data on the prevalence of occupational disease in power plants. I think this would relate again, as would the previous question asked, to the age of the plant and length of duration exposure of power plant workers. There probably is no such thing as "power plant disease;" there are a number of exposures and a number of potential problems. I would not even hazard to guess what the prevalence of disease may be for morbidity or mortality. I would mention some studies that I am aware of, that may be addressing this problem. First of all, Dr. Selikoff at Mount Sinai has done a survey of workers in the Tennessee Valley Authority. I am not aware of this being published anywhere, so I don't know the exact statistics. However, that prevalence survey addressed the prevalence of asbestos-related disease among asbestos insulators who work for the Tennessee Valley Authority. The prevalence of asbestosis by chest radiograph among these asbestos insulators may be similar to other studies of asbestos insulators in New York and New Jersey. The other studies that I'm aware of are those that have been done by the Tennessee Valley Authority. They have been looking at respiratory effects of dusts, particularly coal dust. However, I am very interested in studying power plant workers to see if we can address your question in particular, "What is the prevalence of disease and what diseases are these?"

Dr. Ferris: I gave you, if you recall, the data that were reported. The association from the reanlysis was much, much lower, and it did not reach a level of significance. Those who did the reanalysis felt the observations could have occurred by chance alone. So, on this basis we exclude these as being causally related. I think if there were causality, one would have to be careful about the range over which these observations were made and I think we would all agree that at high levels there is a definite effect of this mixture of SO2 and particulates. But, which is the active ingredient? I don't think we know. I think it very unwise to take data from one level and extrapolate it down to the lower level. This may not be defensible at all. At the levels of pollution we are seeing in most instances around here, there may be a very minimal effect, if any, upon mortality. It may be having an effect upon symptoms but not mortality.

Ray Gordon, West Virginia State Coordinator for EPA: Last May 1978, in Phase I, the ORBES did the first year's interim report. One of the very exciting portions of that presentation involved the Teknekron work. That seemed to indicate the long range transport mechanism and the causative factors for acid rain consideration of a very serious nature. In my role, I've had an opportunity to be approached and deliberate at great length whether, in fact, we should be concerned about the acid rain phenomenon. In addition, since that time, the study indicated that this could be a very large impact area, sort of a funnel effect. Western Pennsylvania, Northern West Virginia, and Eastern Ohio seem to be the receptors of these problems. There have been a number of supportive activities since May 1978. A number of subtle and some very dramatic matters have occurred since the first finding. The EPA office which is not too far from Pittsburgh--in Wheeling, West Virginia--has unoffically observed some phenomenally low pH readings associated with rainfall, in particular with the inversion that occurred last fall, in late October or November, 1978, which was declared to be the worst in the past three years. So it was more than a coincidental relationship. I would like very much to have the panel discuss the acid rainfall. I don't want to reveal the pH readings we get until I hear what you have experienced across the board and particularly the lowest readings that you have seen. We have done some quick literature research and the values we found are startling. I don't see anything in the literature close to what we found. Although it is unofficial data, we have a lot of confidence in it's validity.

Dr. Rom: Acid rains have occurred in the northeastern part of the U.S. This has had some effect on some of the lakes. I do not think we have quite as bad a situation as has occurred in Scandinavia where they had serious effects on lakes such that they had reduction of fish population. I think probably in most of the areas where there are woods or vegetation there is enough buffering capacity to tolerate the acid rains, unless they are at an extremely low pH.

Dr. Stuckel: What's extremely low there?

Dr. Ferris: The low pH's are 3 to 4. Most of this has been attributed to sulfuric acid although the nitric acid probably contributes some. With respect to the alert you talked about, we collected base-line pulmonary function data on the children in Steubenville and followed them during the period of that alert. We are just in the process of analyzing the pulmonary function data to see if it had any effect upon their health. We have not fully completed that analysis as yet.

Dr. Andelman: I think that it would be very difficult to assess health impacts from acid rainfall in this part of the country where we have a lot of acid mine drainage. We certainly have very low pH's in some of the rivers, like the Kiskiminitas or the Monongahela. Sometimes we get pH's as low as three. One question is what is the possible impact of such a lower pH on human health through the route of affecting water treatment or mobilizing trace metals from sediments and things like that. If I had to conjecture, I'd say it's going to be very difficult to assess. What you have to try to do is see if, in fact, you can measure some high levels of metals and if you can do that, then the next step is assessment of the impact on human health. I think that, as Dr. Ferris indicated, probably the greatest concerns about acid rainfall are ecological ones.

Ed Light, Appalachian Research and Defense Fund in West Virginia: I think it's appropriate to make a statement at this point. The key environmental health question ORBES needs to answer is what is the best judgment of dose-response range for sulfate. This conference will be a real disappointment to me as an advisory committee member if information isn't generated to help make this decision. First of all, it appears to be relatively unlikely that S02 direct health effects below standards are significant. Hopefully, but maybe this is too big a hope, EPA will soon get its act together to enforce the existing ambient S02 standard. As a member of another advisory committee on increased coal utilization to the office of Technology Assessment of Congress, I was most impressed by a conclusion of our team of researchers from Harvard School of Public Health under John Spangler on the sulfate question. After a very thoughtful review of all the relevant data, they told us that the epidemiological work on sulfate indicates that some pollutants or combination of pollutants emitted by coal burning power plants poses probable health risks at ambient levels. Increasing emissions will probably increase this risk. Total sulfate isn't necessarily the definite culprit, but it is a valid indicator. They said some of the CHESS findings are not inconsistent with other independent research. Sulfate-effect thresholds concluded by CHESS are basically restated by OTA as its current best estimates, i.e. seven or eight micrograms per cubic meter for asthma aggravation, around twenty-five micrograms per cubic meter for increased mortality. They debunked the common criticisms of epidemiological studies implicating sulfates after a thorough and thoughtful analysis. On smoking, it is important to note that a very high correlation between air pollution and smoking habits of the entire urban population would be required to account for the mortality differences now attributed to air pollution. It is unlikely that this is true. The report generally endorses the Brookhaven approach to estimating the range of sulfate effects. I understand Dr. Hamilton will give detailed discussion of this tomorrow and, in my opinion, this will be the real heart of this environmental health conference. I'd like to make one brief comment on the University of Illinois' unpublished study that was just mentioned. I think this would be a very important contribution to the data ORBES needs to make its decision. It will be helpful to estimate the trends in Chicago sulfate levels and maybe try to work this into the analysis.

Dr. Stuckel: Before there are other questions, are there any statements in response to that?

Dr. Lippmann, New York University: As has been brought out by several speakers and comments at this meeting, there are good reasons not to rush into a sulfate standard. It's quite clear that the bulk of sulfate that we measure most of the time is ammonium sulfate which is innocuous. Sulfuric acid is much more hazardous, but it forms a very variable part of the measured sulfate. While I'm up, if I may, I'd like to respond to something Dr. Hamilton just said about concentrated sulfuric acid depositing in the alveoli. Fortunately, that is physically impossible, primarily because sulfuric acid and other acid droplets are hygroscopic. They can't get to the alveoli in concentrated form even if they're inhaled in the concentrated form, because of the enormous uptake of water vapor as they penetrate through the conductive airways. They have to deposit as very, very dilute droplets.

One thing which the directors of this conference have not addressed is related to the necessity for SOx control. I'd like to refer to something that we saw in a slide projected on the screen yesterday showing haze. Now the SO2, as a gas, does not scatter light. However, when it is oxidized, it forms particles which are acidic. These particles scatter light and produce haze. Haze may be very important, and not only in terms of visibility reduction. It was demonstrated quite effectively by several presentations at a recent Atmospheric Aerosol Conference sponsored by the New York Academy of Sciences in January--that the haze reduces the actinic radiation reaching the ground. Some fairly convincing data is coming to light which indicates that the Ohio River Valley Basin may need to be concerned about the effect of the haze on productivity of crops. I suspect that these considerations, i.e. agricultural productivity, acid rain, and visibility are perhaps the really significant problems resulting from the sulfur oxide emissions in the environment. Perhaps we should deemphasize the search for health effects which may not be there and devote more research to other aspects of the pollution problem which may show effects at lower levels.

Dr. Hamilton: A quick correction, sir. One of the problems of uncertainty is clearly that even when we have a meeting like this, people don't listen to your remarks, or if they do, they mishear them, because I distinctly remember saying that sulfuric acid didn't get neutralized.

Dr. Sati Mazumdar, Department of Biostatistics, University of Pittsburgh: Yesterday and today Dr. Herbert Schimmel's work has been discussed several times. Dr. Schimmel wanted to come to this meeting to discuss a few things and share a few thoughts. Due to personal reasons, he could not make it. As I am quite familiar with his study, he requested that I present his slides and bring out a few highlights of his study. He sent me some reprints of his paper in the Journal of The New York Academy of Medicine. If some of you are interested you can take a copy.

This is the first slide (Figure 2c). His study covers the time period 1963-1976 in New York City. He was investigating the possible association of sulfur dioxide with mortality and also possible associations of particulates with mortality. The time period is 1963 through 1976. He analyzed data for three sub-periods: 1963 to 1969, 1970 to 1976, and the combined period. The average level of sulfur dioxide in the first time period was 0.15 ppm and in the second time period, 1970 to 1976, 0.04 ppm. So you can see there was a multiple reduction in the level of SO2. Now in this first slide there are two sets of figures, one at the bottom and one at the top. The four numbers represented in each time period (each figure) of a set are the magnitude of the mortality variate in the four quartiles. This is the quartile classification of mortality based on SO2. The values, the solid lines, are the magnitude of the adjusted total mortality variate. What Dr. Schimmel wanted to show in this figure, without any numbers, was that using a time series methodology which he developed over ten years, if you only account for seasonal variation, or long term variation (that is what he called filtered only) we see a kind of dose-response relationship that is about the same in the two time periods. So if there is a causal relationship we cannot see it because in the first time period the level of SO2 is very high, about four times higher on the average than in the second time period. He implies there cannot be a causal relationship, if the results for the two time periods are so similar and the levels of SO2 so dissimilar. If we go to the upper set of three figures where the variates are filtered and temperature corrected, that is when the effect of temperature, as well as seasonality on mortality has also been taken into account, we see that all the effects of SO2 on adjusted Total Mortality are nonsignificant. There is no dose response relationship suggested.

The slide (2d) shows the same adjusted Total Mortality where the quartile classification is based on Smoke Shade. Here we see the Filtered and Temperature Corrected set of results. Some association between smoke shade levels and adjusted Total Mortality still remains after correcting for temperature and seasonal variation. The results still suggest a possible dose response relationship between Smoke Shade and adjusted Total Mortality unlike the situation for SO2. Dr. Schimmel has other analyses. This is only one way of looking at the data. In his paper he also presents a two-way classification where he shows that any synergistic effect of the two pollutants is not apparent.

His conclusions from his study are that there is no effect of SO2 on mortality and this is consistent with human and animal experiments where no acute adverse effect is found below 1 ppm.

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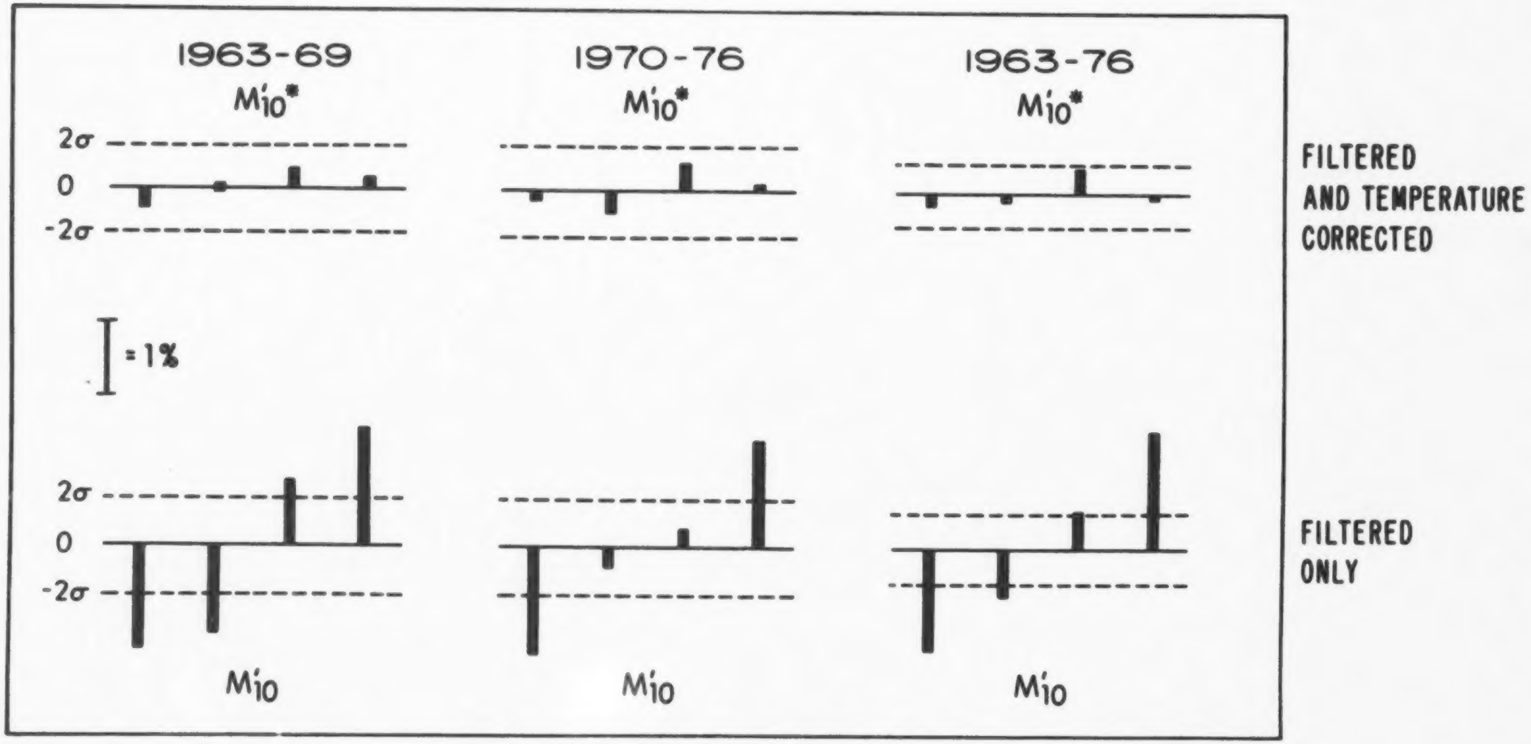

Fig. 2c. M_{0}= Adjusted Total Mortality: Quartile classification based on SO_2.

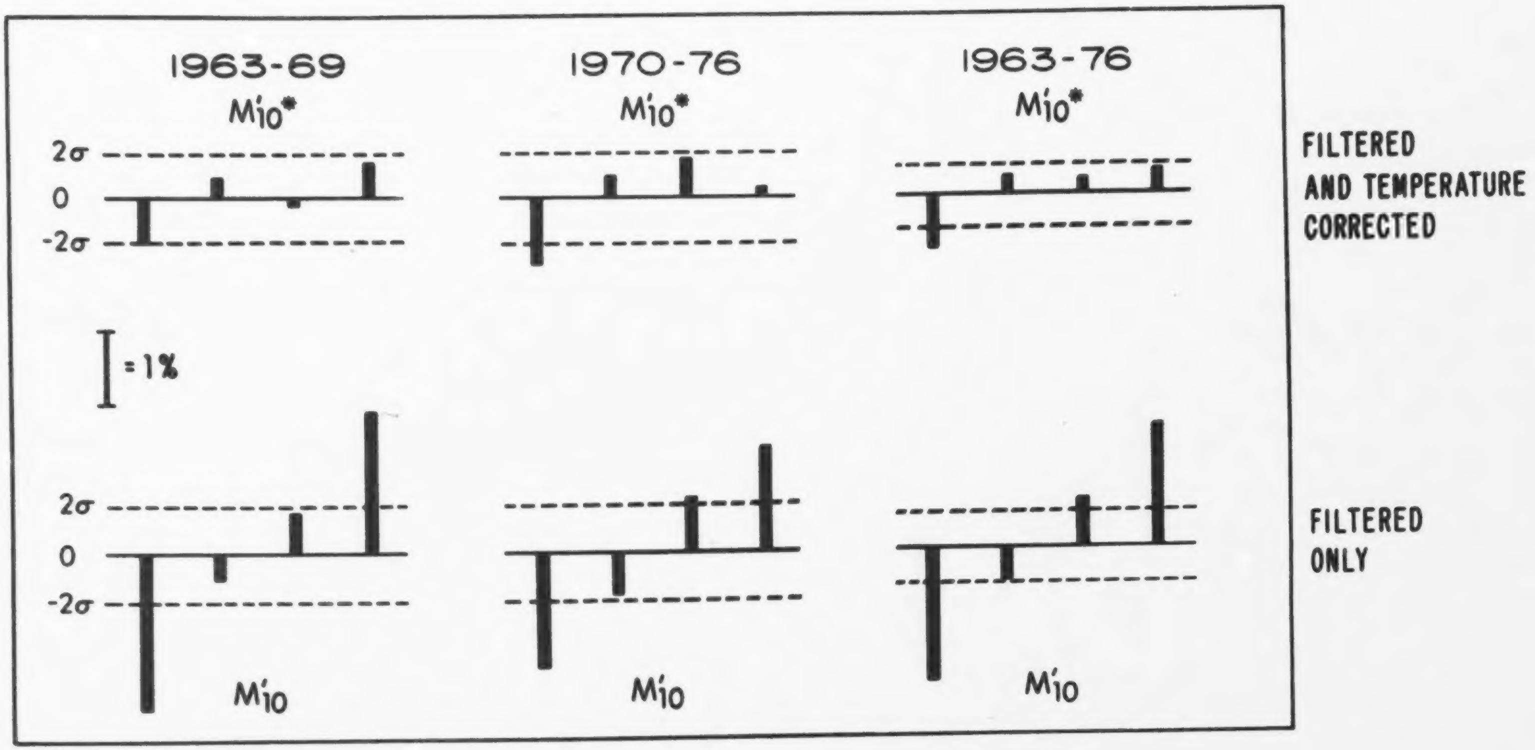

Fig. 2d. M_{10}= Adjusted Total Mortality: Quartile classification based on Smoke Shade.

SESSION IV: HEALTH ASPECTS OF TRANSPORTATION AND TRANSMISSION

Tuesday afternoon, March 20, 1979

Moderator: Hugh T. Spencer, Ph.D.
Professor of Chemical Engineering
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HEALTH ASPECTS OF FUEL AND WASTE TRANSPORTATION
By
Samuel C. Morris, Ph.D.

HEALTH ASPECTS OF POWER TRANSMISSION
By
Richard D. Phillips, Ph.D.

HEALTH BENEFITS AND RISKS OF ELECTRIC POWER CONSUMPTION
By
Ronald E. Wyzga, D.Sc.

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HEALTH ASPECTS OF FUEL AND WASTE TRANSPORTATION

By

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INTRODUCTION

Energy resources are frequently not found where we could like them to be. We thus expend considerable effort in transport. Living on Long Island, I am reminded of the cost involved every time I buy gasoline for my car or oil for my furnace. I do not find much solice in the knowledge that 200 years ago, when we were on a wood economy, the area on Long Island where I live was a major supplier of energy to New York City. As a sidelight, if you have ever wondered what happened to all the horse manure generated in New York back then, it was sent to Long Island on the same boats that brought wood to the city. Unfortunately, today we have not been able to find a profitable return cargo for most of our energy transport, so the round trip cost, in dollars, health and environmental impact, must be charged against the transported fuel.

What are the health costs of energy transport? Accustomed as we are to the carnage on our highways, we should not be surprised that there are some. In the U.S. we move around annually about 6.5×10^8 tons of coal, over 4×10^9 barrels of crude oil and over 5×10^9 barrels of refined products, and over 2×10^{12} cubic feet of natural gas. Most health damage is due simply to the trauma resulting from accidents which accompany the handling and movement of such a large mass of material. Some of the health risk, however, stems from the unique nature of the fuels transported. Table 1 summarizes estimates of the health damage of energy transport in several fuel cycles on a unit energy basis. Since coal has the highest health risk and nuclear the most controversial, I will go through these two in some detail.

Table 1
Health Damage in Energy Transport
per GW_e-yr

Fuel Cycle	Deaths	Injury	Latent Cancer
Coal	0.5	2	-
Oil	0.08	7	-
Natural Gas	0.004	1	-
LNG	10^{-6} to 10^{-1}		
Nuclear	0.02	0.3	0.002

COAL TRANSPORTATION

Coal is transported by rail, water (rivers, Great Lakes, and intercoastal waterways), truck, and/or conveyer belt. One slurry pipeline is in operation and pipelines are a potentially significant mode of coal transport in the future.

Most coal moves by rail. Coal constitutes 29% of all rail freight in the U.S. and 13% of all freight revenue. By comparison, coal transport makes up 20% of all rail freight in the USSR. Coal is moved in hopper cars which are unloaded by turning the car itself over, by opening bottom hoppers, or, more rarely, by unloading from the top. The cars are generally loaded at mine sidings or tipples which collect from several mines. The average hopper car carries 75 tons of coal, older cars 55 tons and newer ones 100 tons. Size of hopper car used depends on the condition of the track. Modern unloading facilities are frequently trestles running over storage piles into which coal is dumped from moving cars. Many have loading and unloading rates of thousands of tons per hour.[1]

Statistics are not maintained for accidental deaths and injury in railroad transportation by type of freight carried; thus, it is not possible to determine directly from available data the mortality effects of transporting coal by rail. There are several ways to estimate this value. The simplest is to assign a proportionate share of all railroad deaths to coal transport. This apportionment may be on the basis of total weight or bulk of material transported, train-miles traveled, or ton-miles traveled. Over 60 percent of accidental deaths associated with freight traffic result from rail-highway grade crossing accidents, primarily due to collisions between trains and motor vehicles.[2] The number of intersections crossed and the characteristics of each crossing (e.g., highway vehicle and train traffic density, type of crossing protection) are crucial parameters. Several models have been developed to predict the accident risk at grade-crossings based on such factors. Rogozen, et al.,[3] use such a model to estimate the impact of specific unit train routes compared to coal slurry pipelines. They conclude that an increase in train traffic is non-linearly related with statistics such as train-miles or ton-miles. In a typical example, as the average number of trains per day increased by 70%, the predicted accidents increased by only 30%. While there are many factors which cannot be considered in these models, it seems clear they offer the best approach to quantifying impacts for particular cases. A great deal of information is required, however. The routes to be followed must be specified; for each grade-crossing on route, the following data is needed: average daily highway traffic, average daily train traffic, type of crossing protection, urban or rural location, and single or multiple tracks. For broad assessment purposes in which specific routes are unknown, a simpler approach is required. Estimates below are based on ton-mile and train-mile apportionment. Since the detailed models indicate a non-linear response, these are average, rather than marginal, estimates. Examples cited by Rogozen, et al., suggest the marginal estimates may vary downward from the average by as much as a factor of 5. Since coal represents a substantial share of all rail transport, and an even greater share in areas with heavy coal traffic, the average figure may be reasonable when estimating effects of total U.S. coal transport. When estimating the incremental effect of a single plant or a perturbed scenario, a marginal estimate would be desired. All grade-crossing accidents involving trains have been charged against coal transport in calculations made below. One might argue that since the fault frequently lies with the motor vehicle, that only a portion (and perhaps a small portion) should be charged to coal transport.

There is some reason to believe that unit coal trains may create different exposure situations than the average freight train. Coal has a greater density than average freight, coal cars are generally bigger and heavier and coal trains longer than average. Train velocity may be different. Unit trains generally operate on well used and maintained lines and it has been suggested that they may be less likely than average freight trains to pass non-signal crossings. Concerning the 40 percent of accidents not at grade-crossings, unit trains generally can avoid hazardous switching and yard operations. While one might suspect that unit trains are less likely to be involved in fatal accidents than are average freight trains, there is no quantitative basis in available data on which to base an adjustment. Occupational accidents associated with unit trains seem likely to be concentrated at on- and off-loading facilities.

Schoppert and Hoyt, in a sample of grade-crossing accident reports from six states, found that grade-crossings have a turbulent effect on motor vehicle traffic resulting in accidents not involving a train.[4] Accidents not involving trains were twice as frequent as accidents involving trains and motor vehicles. Of the accidents not involving trains, half occurred when a train was present but not involved and half when a train was not even present. Based on detailed data from 16,000 crossings in Illinois, it was found that the fatality rate was much higher in accidents involving trains but the non-fatal injury rate was higher in non-train crossing accidents (Table 2). These accidents

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Table 2
Accidents and Health Damage at Railroad Grade-Crossings by Train Involvement[(a)]

Train Involvement	Accidents		Deaths		Injuries	
	No.	%	No.	Per Acc*	No.	Per Acc*
Train Involved	1113	30.7	290	0.26	738	0.66
Train Not Involved	2514	69.3	25	0.01	1707	0.68
Total	3627	100.0	315		2445	

(a) Based on Illinois data for 1962-1964. From Schoppert and Hoyt, National Cooperative Highway Research Program Report 50, 1968, pp. 20-21.

* Accidents

not involving trains have not been included in calculations below. In cases where a crossing exists solely for the benefit of a unit train such accidents should be charged against coal transport to the same extent as accidents involving trains. In the majority of cases, however, where crossings have mixed use, it is not clear to what extent non-train accidents should be charged against coal transport. Lorber has extensively reviewed Schoppert and Hoyt and developed a statistical model of grade-crossing accidents based on their data.[5]

In unit trains, and for the most part in non-unit trains also, the cars must return to the mine empty. Thus, train mileage refers to the round-trip distance per trip times the number of trips while ton-mileage is calculated as tonnage hauled per trip times one-way distance (no ton-miles are incurred on the empty return trip) times the number of trips.

There were 6.95×10^{12} ton-miles of freight movement on U.S. railroads during the period examined, 1966-74. Freight trains logged 3.97×10^{9} train-miles.[6] Statistics for total deaths and injuries, deaths and injuries among employees and among the public are given in Table 3 per ton-mile and per train-mile.

The basic data from which the statistics are calculated are given in Table 4. The annual statistics show a slight downtrend in public deaths and injuries.* Given that both railroad and

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*The 1973 deaths and injuries among the public seemed anomalously low. The data for 1973 were rechecked with DOT to assure they were correct (personal communication with M. Snellings, 6/8/78).

Table 3
Deaths and Injuries Associated with Railroad Freight
Operations per Ton-mile and Train-mile

	PUBLIC				EMPLOYEES			
Year	Deaths per 10^9 ton mi	Deaths per 10^6 train mi	Injuries per 10^9 ton mi	Injuries per 10^6 train mi	Deaths per 10^9 ton mi	Deaths per 10^6 train mi	Injuries per 10^9 ton mi	Injuries per 10^6 train mi
1966	2.16	3.65	4.30	7.25	0.050	0.085	5.33	9.00
67	2.08	3.55	4.47	7.65	0.060	0.102	4.41	7.55
68	1.98	3.43	4.26	7.38	0.059	0.103	5.13	8.88
69	1.95	3.45	4.50	7.97	0.072	0.127	4.94	8.75
70	1.95	3.50	3.86	6.90	0.077	0.138	4.50	8.06
71	1.94	3.33	4.05	6.97	0.041	0.070	3.89	6.69
72	1.82	3.14	3.99	6.87	0.048	0.082	3.56	6.14
73	0.38	0.68	0.45	0.81	0.068	0.124	3.54	6.44
74	1.62	2.95	3.56	6.45	0.055	0.100	4.02	7.28
66-74	1.74	3.05	3.66	6.42	0.059	0.103	4.44	7.78

data from Table 4.

Table 4
Railroad Freight Statistics 1966-74

			PUBLIC		EMPLOYEE	
Year	Ton mi x 10^9	Train mi x 10^6	Deaths	Injuries	Deaths	Injuries
1966	738	437	1595	3173	37	3938
67	719	420	1493	3214	43	3783
68	744	429	1474	3168	44	3814
69	768	433	1496	3455	55	3790
70	765	427	1495	2949	59	3443
71	740	430	1432	2994	30	2874
72	777	451	1414	3099	37	2767
73	852	469	321	381	58	3019
74	851	469	1383	3027	47	3418
66-74	6954	3965	12103	25460	410	30846

Ton-mile and train-mile data from Yearbook of Railroad Facts, Association of American Railroads, Washington, DC, 1977, pp. 29 and 37.

Injury and death data from U.S. Dept. of Transportation, Federal Railroad Administration, Office of Safety, Accident Bulletin Nos. 135 through 143, 1966-74. Data for 1975 and later years are not available in this form.

automobile traffic is increasing, this must reflect the effort of the railroads, state highway departments, and Department of Transportation to reduce accidents, particularly at grade-crossings. Statistics were not calculated later than 1974 since accident data were no longer reported separately for freight traffic by DOT after that year.

The average haul length for the industry as a whole is over 500 miles,[6] but coal shipments have an average haul length of 300 miles.[7] Assuming 1 GW_e-yr electrical output requires 3.4×10^6 tons of coal, this yields 1×10^9 ton-miles.

Assuming an optimum unit train size of 15,000 net tons, annual round trips required would be $\frac{3.4 \times 10^6 \text{ tons}}{1.5 \times 10^4 \text{tons/train}} = 227$ train trips.

227 trips x 600 miles round trip = 1.4×10^5 train miles per GW_e-yr.

Non-unit train movement would involve more train miles to deliver the same amount of coal, but only a share of the effort can be **charged** to coal transport. If one takes these results and applies the average figures over the 1966-74 interval from Table 2:

	cases per GW_e-yr	
	Train-mile basis	Ton-mile basis
public deaths	0.41	1.8
public injuries	0.86	3.8
employee deaths	0.014	0.06
employee injuries	1.0	4.6

The estimates from the train-mile approach give a closer fit with the results of the more detailed approach in Rogozen when compared in actual cases.[3] For haul distances other than 300 miles, the estimates are linearly proportional to changes in haul distance. Note that accidents involving the public, mostly collisions between motor vehicles and trains, have a high death to injury ratio, while occupational accidents involve many more injuries than deaths.

Since the bulk of the effects involve collision with automobiles, one would expect variation by region due to differences in the density of automobile traffic and driving patterns. Table 5 provides estimates of the variation in accident rates by railroad district. These tables combine data on all rail traffic, both freight and passenger. The difference in deaths per train-mile among regions is less than a factor of 2 and in injuries less than ± 10%.

Potential health effects not quantified result from the contribution of diesel exhaust from coal unit train locomotives to air pollution and to the possible impairment of emergency medical service in communities where major streets are frequently blocked by unit trains.

Barge transport of coal is estimated to be an order of magnitude less hazardous than rail transport and slurry pipeline (based on experience with oil) a further order of magnitude safer.[8]

Table 5
Casualties by Railroad District 1976
(Class I Railroads, Line-Haul)

	Eastern	Southern	Western
Train Miles x 10^6	276.4	118.4	329.4
Train Accidents-Class I Line-Haul			
killed	56	28	68
injured	464	345	425
Train Incidents-Class I Line-Haul			
killed	416	225	575
injured	4138	2413	5498
Non-train Incidents-Class I Line-Haul			
killed	46	84	87
injured	18990	7141	19346
Total All Casualties			
killed	518	397	730
deaths/10^6 train-miles	1.87 (1.7-2.0)*	3.35 (3.0-3.7)	2.22 (2.1-2.4)
injured	23592	9899	25269
injuries/10^6 train-miles	85.4 (84.3-86.5)*	83.6 (82.0-85.3)	76.7 (75.7-77.7)

Source of data is for Class I railroads, line-hauls from USDOT, Federal Railroad Administration Accident/Incident Bulletin No. 145, pp. 64, 68 and 70.

*Range in parentheses is 95% Poisson confidence interval.

Truck transport has been the most rapidly growing coal transport mode. Twelve percent of total coal production, or about 8 x 10^7 tons, is delivered by truck. Haul distance averages 50 to 75 miles.[9] This does not include truck-haul of coal to tipples for loading on rail or barges. Assuming a 30 ton load of coal per trip, 3.4 x 10^6 tons requires 1.1 x 10^5 trips. For a 100 mile round trip, this yields 1.1 x 10^7 vehicle-miles. Common carrier truck fleets average 7 accidents per 10^6 vehicle miles, resulting in 79 accidents per GW_e-yr. Assuming 0.03 deaths and 0.5 injuries per accident results in 2.4 deaths and 40 injuries per GW_e-yr.

The principle waste products in the coal fuel cycle subject to transport are fly ash and scrubber sludge. In general, these wastes are maintained on or near the plant site to avoid unnecessary transportation. Current designs use slurry pipelines which are expected to produce minimal health effect.

NUCLEAR FUEL CYCLE

Transport is much more complex in the nuclear fuel cycle than for coal because there are many more steps in the cycle. The mass of nuclear materials required per GW_e-yr is much less than for coal, however. Even with the elaborate precautions required in the shipment of nuclear materials, transport is a minor part of the cost of entire fuel cycle. Because of this, other factors predominate in processing facility location leading in some cases to cross flows of material or longer hauls than would otherwise be required.[10] Figure 1 illustrates the transport steps in the full nuclear fuel cycle. These are detailed in Table 6 and its accompanying notes for the steps needed to support 1 GW_e-yr production of electricity in the nuclear fuel cycle the way it is currently operated in the United States, although including shipment of spent fuel assemblies to interim storage. Later introduction of a federal waste repository would require an additional 7 trips by rail from interim storage to the repository.

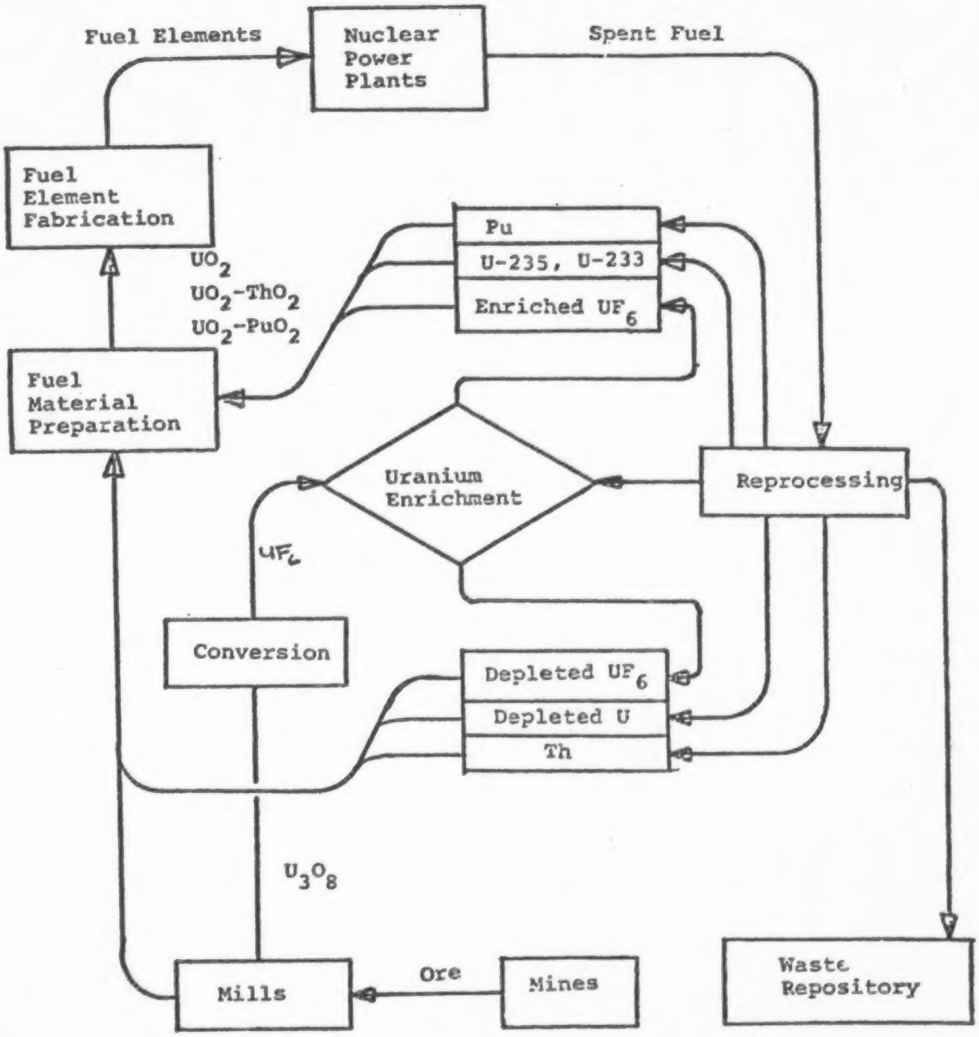

FIGURE 1 Full Nuclear Fuel Cycle

TABLE 6. NUCLEAR FUEL CYCLE TRANSPORT (NO RECYCLE)

per GW_e-yr

Material	Form	From	To	Mode	QUANTITY SHIPPED		DISTANCE SHIPPED	Number of Shipments
					Mass	Activity	Miles	
ORE	Solid Bulk	Mine	Mill	Truck	1.7x10^{5}MT$^{(a)}$	1.4x10^{3}Ci$^{(b)}$	0-40 (5)$^{(c,d)}$	6300$^{(e)}$
Concentrated U_3O_8("yellow cake")	Granular or powder in 55 gal steel drums	Mill	Conversion Plant	Truck	340 MT$^{(f)}$	360 Ci$^{(g)}$	275-2200(1000)$^{(h)}$	20$^{(i)}$
UF_6	Solid in 10 to 14 ton cylinders	Conversion Plant	Enrichment Plant	Truck	394 MT$^{(j)}$	62$^{(k)}$	20-900 (500)$^{(l)}$	22$^{(m)}$
Enriched UF_6	Solid in$^{(n)}$ 2.5 ton cylinders	Enrichment Plant	Fuel Preparation Plant	Truck	64 MT$^{(o)}$	62$^{(p)}$	0-1600 (750)$^{(q)}$	11$^{(r)}$
UO_2	Powder or$^{(n)}$ pellet in steel pails supported in 55 gal drums	Fuel Preparation Plant	Fabrication Plant	Truck	49 MT$^{(s)}$	62$^{(t)}$	0-1600 (750)$^{(u)}$	12$^{(v)}$
Unirradiated Fuel Assemblies	Type B Containers	Fabrication Plant	Reactor	Truck	43 MT U$^{(w)}$	62$^{(x)}$	(1000)$^{(y)}$	7$^{(z)}$
Irradiated Fuel Assemblies	Type B Containers	Reactor	Storage	Truck Rail	16 MT $^{(aa)}$ 24 MT $^{(aa)}$	7.6E6 1.1E7	(500)$^{(bb)}$ (1000)$^{(bb)}$	20$^{(aa)}$ 5$^{(aa)}$
Low Level Waste		Conversion Plant	Burial Site	Truck	280 M$^{3(cc)}$		20-900 (500)$^{(dd)}$	25-38$^{(ee)}$
Low Level Waste		Fabrication Plant	Burial Site	Truck	180 M$^{3(ff)}$		20-900 (500)$^{(dd)}$	19-25$^{(ee)}$
Low Level Waste	Solids packed in drums	Reactor	Burial Ground		100-1000 m^3 $^{(gg)}$	2E3 Ci$^{(hh)}$	(500)$^{(ii)}$	60$^{(jj)}$

Notes to Table 6:

a. Calculated from GESMO, p. IV J (E)-17. 1.77E6/(4732 x 1.1) = 340. MT U_3O_8. An ore grade of 0.2% this yields 1.7E5 MT ore. Note WASH-1238 & WASH 1248 (p. H-7) have a much lower fuel requirement since they assume recycle and only 0.8 GWe-yr.

b. Calculated from GESMO p. IVH 14, 4.51E5 x 14 = 6.31E6 ci 1975-2000 total from GESMO p. IVJ (E) 17, 6.13E6/4.732E3 = 1.33E3 ci/GW$_e$-yr.

c. WASH-1248, p. H-7.

d. Number in parenthesis is the distance used in the models of WASH 1248 and GESMO.

e. Scaled from WASH 1248, p. H-7, 182 MT U_3O_8 and 3350 trips. (340/182 x 3350 = 6258.

f. From GESMO, p. IV-J (E) 17, 1.6E6/(4732 x 1.1) = 307 MT U_3O_8. Assuming yellowcake is 90% U_3O_8, 307/0.9 = 341 MT yellowcake.

g. Following calculation was provided by J. Nagy. The activity of a mass of radionuclide is given by:

$$C\ [Ci] = 3.5768 \times 10^{11} \frac{M}{AT} \quad \frac{[\text{Mass (metric tons)}]}{[\text{atomic wt}]\ [\text{half life yrs}]}$$

For Unit Mass

Nuclide	U-235	U-238
M	1	1
A	~235	~238
T	7.04×10^8	4.468×10^9
C [/tonne]	2.162	0.336

The activity of nuclear fuel is given by:

$$2.162\ f_{235}\ d_{235} + 0.336\ f_{238}\ d_{238} \quad \text{ci/tonne}$$

where,

f = parent nuclide fraction by weight

d = number of main sequence daughters in equilibrium plus parent f and d depend on before/after milling and before/after enrichment. Rough values are given below:

	f_{235}	d_{235}	f_{238}	d_{238}	activity (Ci/MT-u)
before milling	0.007	11	0.993	14	4.84
	0.197 (4.0%)		4.671		
after milling/ before enrichment	0.007	2	0.993	4	1.36
	0.030 (4.0%)		1.335		
after enrichment	0.032	2	0.968	4	1.44
	0.138 (9.6%)		1.301		

h. WASH 1248, p. H-9.

i. Scaled from WASH 1248, p. H-9, $\frac{307}{182} \times 12 = 20$.

j. From GESMO, p. IV-J (E) 17, 1.21E6/(4551 x 1.1) = 266 MT U. or 340 MT UF_6.

k. See note g.

l. WASH 1248, p. H-10.

m. WASH 1248, p. H-10.

n. Shipped as Class A, fissile material (NUREG-0170, p. A-6)

o. Calculated from GESMO, p. IV-J (E) 17, 1.89E5/4346 = 43 MT U or 64 MT UF_6.

p. See note g.

q. WASH 1248, p. H-12.

r. Scaled from WASH 1248, p. H-12

$$(64/52) \times 5 = 6.2$$

s. See note o. 43 MT U = 49 MT UO_2.

t. See note g.

u. WASH 1248, p. H-14.

v. WASH 1248, p. H-14, 40 MT UO_2 requires 9 Shipments.
(49/40) x 9 = 12.3

w. See note o.

x. See note g.

y. GESMO, p. IV-G 4.

z. From GESMO p. IV-G 4 - 30,800 shipments
From GESMO p. IV-F (E) 17 4346 GW_e yr equivalent 30,800/4346 = 7

aa. From GESMO, p. IV-G 4
50,200 MT Uranium + Plutonium by truck in 61,400 shipments
75,200 MT Uranium + Plutonium by rail in 15,300 shipments
From GESMO, p. IV-F (E) 17 3072 GW_e yr equivalent.

bb. GESMO, p. IV-G 4.

cc. From WASH 1248, p. H-13, Scaled to 1 GW_e yr 8000/0.8 = 10^4 ft^3 = 280 m^3

dd. See note cc.

ee. See note cc.

ff. See note cc 5000/0.8 = 6250 ft^3 = 180 m^3.

gg. USEPA, Environmental Analysis of the Uranium Fuel Cycle (EPA-520/9-73-003-B, Part I, 1973, p. 138 gives 3000 - 55 gal. drums (620 m^3). WASH-1238, p. 49 gives 3800 ft^3 (100 m^3). The SDG and E Environmental Report for Sundesert Nuclear Plant Fig. 3.5-4, gives 300 - 55 gal. drums plus 483-50ft^3 containers (1000 m^3).

hh. WASH-1238, p. 49.

ii. EPA 520/9-73-003-B, p. 139.

Introduction of reprocessing and recycling, of either uranium or of uranium and plutonium introduces some additional transport steps, but reduces the flows in others. The differences are estimated in detail in GESMO.[11]

Although the quantities shipped are much lower than in the coal fuel cycle, there is greater concern due to the intrinsic risk of exposure to radiation both routinely and in accidents. Workers involved in transport are not classed as radiation workers and not subject to individual monitoring. Thus, occupational exposures of transport workers cannot be obtained directly from available data. A recent surveillance program showed that some transport workers receive a higher annual dose than permissable for the general public.[12] As a result, there are plans to require individual exposure monitoring for certain classes of transport workers.[13]

Dose calculations have been made in several reports based on assumed dose rates and time spent by workers at various locations. Table 7 summarizes the collective radiation dose and the expected latent cancer fatalities associated with transportation between various stages of the nuclear fuel cycle. The cancer estimates, based on health damage functions developed by the BEIR committee,[14] are very small; the total estimated exposure in all transport for 1 GW_e-yr might lead to 1 cancer in 1000 years. In 1975, fuel cycle related transport contributed less than 10% of the population dose from transport of all (non-military) radioactive materials. This is expected to grow to 15% by 1985.[15] A more

TABLE 7

ROUTINE RADIOLOGICAL RISKS OF TRANSPORT IN THE NUCLEAR FUEL CYCLE

MATERIAL	POPULATION	MAN-REM PER GW_E-YR	LCF*
ORE	WORKERS	0.06	E-5
INTERMEDIATE MATERIALS **	WORKERS	0.23	4E-5
	PUBLIC	0.04	7E-6
UNIRRADIATED FUEL	WORKERS	0.01	2E-6
	PUBLIC	0.001	0.2E-6
IRRADIATED FUEL	WORKERS	1.3	2E-4
	PUBLIC	2.1	3E-4
LOW LEVEL WASTE FROM REACTOR	WORKERS	1.0	2E-4
	PUBLIC	1.2	2E-4
TOTALS	WORKERS		4E-4
	PUBLIC		5E-4
	TOTAL		9E-4

*LATENT CANCER FATALITIES.

**INCLUDES U_3O_8, UF_6, UO_2 AND LOW LEVEL WASTES FROM "FRONT END" OF FUEL CYCLE.

BASED ON WASH 1238, P. 8 AND WASH 1248, P. H-21. ADJUSTED FOR NO-RECYCLE AND SCALED TO 1 GW_E-YR.

important concern is the risk of larger potential exposure in the event of an accident. Both the Nuclear Regulatory Commission and the Department of Transportation have promulgated regulations aiming to minimize the chance of an accident involving a radiation release and minimizing the impact were a release to occur. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) concluded that the annualized exposure from transport accidents is an insignificant addition to normal transport risk.[16] A WHO working group examined the risk of spent fuel accidents and reported that, "as the fuel assemblies are stored for extended periods before shipment, most of the radioactivity will be due to the longer lived strontium-90 and caesium-137, which are not volatile.....in an accident causing rupture of the shipping container, any radioactive contamination occurring would be localized. Although in extreme cases evacuation of people in the immediate vicinity might be desirable, more general evacuation, although common in accidents including other toxic materials such as noxious gases, would not be necessary."[17]

The most extreme case would be an accident involving a large release of radioactive material in a high population density area such as New York City or Chicago.

Table 8
Results of Low Probability/High Consequence Accident Analysis

Material	Latent Cancer Fatalities	Early Morbidities	Early Fatalities
Spent Fuel ($3000/2.17 \times 10^5$ ci)*	10	6	0
Plutonium (1.13×10^6 ci)**	3964	952	18

*Truck shipment through NewwYork City. 3000 dispersible/2.17×10^5 non-dispersible.

**New York City air cargo of overseas fuel. 100 kg of PuO_2, 100% released, 5% aerosolized.

From A.R. DuCharme, et al., SAND77-1927, pp. 118-120.

Taylor, et al., in a study of potential accidents in New York City found that time of day and routing restrictions significantly affect accident impacts and that buildings significantly limit radiation exposure.[18] In a later, more detailed report, the largest domestic fuel cycle related accident (spent fuel) examined was calculated to produce only 10 latent cancer fatalities (Table 8).[19] Urban accidents involving recycled plutonium have been estimated to result in less than 100 latent cancer fatalities.[20]

Although the transport of fuel cycle materials prior to enrichment has generally assumed to pose insignificant radiological hazard, a recent accident in Colorado (September 1977) involving a spill of several tons of yellow cake resulted in extensive clean-up and review of possible regulatory action covering future shipments by NRC and DOT, and has been cited in a recent HEW report as one example raising pressing questions on the need for emergency response planning.[25]

Finally, transport of nuclear material also involves the common accident risks faced by coal. The shipments detailed in Table 6 sum to over 300,000 vehicle-miles per GW_e-yr, including return trips. If fatality and injury rates are 5×10^{-8} and 8.7×10^{-7} respectively,[22] expectation is 0.02 deaths and 0.2 injuries per GW_e-yr.

SUMMARY

Coal transport accidents raise almost no concern, yet can involve more loss of life than any other step in the fuel cycle. Perhaps the reason is that the accidents are common, every day auto accidents - or that the biggest imaginable catastrophy would be a school bus being hit by a train. Yet perhaps the level of attention given this subject is appropriate to the level of risk. If one were concerned with grade-crossing accidents, they would be better addressed in the whole, rather than restricting interest to accidents involving coal trains. In contrast, transport of nuclear fuel cycle materials raises widespread concern - despite the fact that all assessments show minimal effects - order of magnitude less than the corresponding effects from coal transport. Even the largest calculated accidents in nuclear transport have a lower cost in life than the annual damage from coal transport, although the clean-up costs in nuclear transport accidents can approach $\$10^9$. It is interesting to note that despite the low risk of radiologic exposure, the nuclear fuel cycle involves more vehicle-miles of travel than coal. Injury from common accidents is estimated to be lower in the nuclear cycle only because of the lower accident rate associated with the transport of hazardous materials.

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HEALTH ASPECTS OF POWER TRANSMISSION

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Health Aspects of Power Transmission

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There has been a rapid growth of power transmission systems in the United States over the last several decades and this growth is expected to continue. There are currently more than 40,000 circuit miles of overhead ac (60-Hz) transmission lines in the United States operating at extra-high-voltage (EHV), 345,000 to 765,000 volts, and many more miles are planned over the next several years. In addition, ultra-high-voltage (UHV) transmission systems are now being developed that will operate at 1,100,000 to 1,500,000 volts.

Considerable controversy has developed over the past several years concerning the health aspects of existing EHV and proposed UHV transmission lines. Several scientists and concerned groups claim that the electromagnetic fields in the vicinity of these power lines produce adverse biological effects and are hazardous to man (*1,2*). Other scientists state that such claims lack scientific support (*3,4*). Legal proceedings have been unable to resolve this issue because of the lack of reliable data. In recognition of the need for scientifically sound data to make a risk assessment and to insure public health and safety, research programs have been initiated by the Department of Energy (DOE) and Electric Power Research Institute (EPRI) to assess the health aspects of power transmission systems.

Four research projects are in progress at Battelle Northwest as part of the DOE and EPRI programs. In one of the DOE projects, a multidisciplinary research team, consisting of physical and biological scientists, is conducting a broad and comprehensive study to screen for effects on rats and mice of acute and chronic exposure to 60-Hz electric fields. In a companion project, supported by EPRI, miniature swine are being exposed chronically to a 60-Hz electric field over two generations to assess possible effects of exposure on clinical parameters and on reproduction, growth and development. A third project is examining the potential of static (dc) and 60-Hz electric fields to induce genetic changes in fruit flies and bacteria. The fourth project is evaluating the ecosystem in the vicinity of a 1,100 kV test transmission line at Lyons, Oregon.

The small and large animal studies were started in 1976 and a large amount of data has been generated from these projects during the past three years (*5-11*). This paper will summarize the results of the small animal project and relate our findings to the results of other studies reported in the literature.

EXPOSURE SYSTEMS

The environment in the vicinity of high voltage transmission lines is complex and includes many factors that might be sources of biological effects (Table 1). For the purpose of controlled laboratory studies we chose to retain only those which dominate in the transmission line environment and which, based on existing literature, have been reported by some laboratories as having produced effects. This decision led to system design criteria which eliminated or minimized all the factors in Table 1 except the external electric field and the unavoidable consequences of physical interaction between this field and the exposed subject (internal fields and currents; field perception).

Table 1. Possible Electromagnetic Field-Related Sources of Biological Effects in the Vicinity of ac Transmission Lines

Primary Field Effects	Secondary Factors
Large External Electric Fields	Spark Discharges
Small Internal Electric Fields	Steady-State Currents
Electric-Field-Induced Body Currents	Corona Discharge Ozone Audible Noise Radiofrequency Fields
Small External and Internal Magnetic Fields	Hum and Vibration (of significance mainly in laboratory simulations of the transmission line fields)
Magnetic-Field-Induced Body Currents	Higher-Frequency Harmonics in the Electric and Magnetic Fields
Field Perception Hair Stimulation Other Modes?	

The maximum electric field encountered by man under EHV transmission lines is about 9-12 kV/m. The currents and electric fields induced within the body are a function of the field intensity and the body shape. Values are quite different for man (biped) than those for rodents and swine (quadrupeds). Accordingly, considerable care must be exercised in designing animal experiments and using data from such studies to predict possible effects on humans in the same situations. This is illustrated in Figure 1, which shows a man, a pig, and rat placed in a uniform 60-Hz electric field of 10 kV/m. The strongest surface electric field generally occurs at the top of the body and is enhanced over the uniform field value by a factor of 15-20 for man (*12*), 7 for the pig (*13*) and about 4 for the rat (*11*). Estimated current densities are shown at two locations within each body, the trunk and lower limbs, and illustrate the strong dependence on body shape. The total current between each body and ground, the short-circuit current (I_{sc}), is also shown. Based on these data, a much higher unperturbed field would have to be used with pigs or rats to produce surface fields and internal current densities comparable to those in a man exposed to 10 kV/m. Scaling factors between rat and man are: enhancement, ∿ 4:1; average trunk current density, ∿ 15:1; average current density in the lower limbs, ∿ 5:1. Comparable scaling factors between pig and man are about 2.5, 7 and 2.5, respectively.

We selected 30 kV/m for the pig exposures and 100 kV/m for the rat and mouse exposures in the biological experiments. Assuming a scaling factor of 3 between pig and man, and a factor of ∿ 10 between rat and man, the field strengths used in the experiments would be equivalent to exposing man to about 10 kV/m.

Three systems were built for exposing small laboratory animals to uniform, vertical, 60-Hz electric fields: a rat exposure system, capable of simultaneously exposing 144 rats individually housed in plastic cages; a mouse exposure system in which 288 mice, 3 per cage, can be exposed simultaneously; and a special test system for experiments requiring direct data acquisition from individual or small numbers of animals during or immediately after exposure. The same numbers of rats and mice are sham-exposed under environmental and housing conditions identical to those of the exposed animals.

One of the four racks used for exposing rats is shown in Figure 2. Each rack consists of four electrodes, with the rats individually housed in plastic cages located in the three lower electrodes. The animals are in electrical contact with a metal mesh floor that forms the top layer of each two-layered electrode. Animal excreta falls through the mesh floor onto absorbent paper in a field-free region on a lower metal plate. The water system is located in this space between the mesh floor and the lower plate, with a small demand-type water nozzle located at the floor level of each cage. This design eliminates possible perturbation of electric field uniformity by the watering system, eliminates shocks to the animals during drinking, and minimizes mouth-to-nozzle steady-state currents during drinking. Pellet food is provided ad libitum in slot hoppers at the front of each cage. The profile of the food delivery system is low to minimize any potential perturbation of field uniformity by the food.

The mouse exposure rack (not shown) is identical to the rat exposure rack except for a lower cage height, a small vertical separation between electrodes, and an additional electrode.

Extensive measurements have been made to characterize the three small animal exposure systems. Electric field uniformity is ± 4% over the total cage area in the rat and mouse exposure systems. The exposure systems are free of detectable levels of audible noise and ozone, and there is very little harmonic distortion. Animals do not receive spark discharges or other shocks from the housing system or from other animals during exposure to electric fields.

Photographs of the system used to expose miniature swine to a vertical, 60-Hz electric field are shown in Figures 3 and 4. Exposed and sham-exposed pigs are housed in separate, environmentally equivalent buildings (Figure 3). The overhead electrode and individual animal stalls are shown in Figure 4. Electric field uniformity over the area of a stall is about ± 2.5%. No levels of audible noise, hum, vibration or ozone have been detected with the high-voltage system operating.

BIOLOGICAL EXPERIMENTS

Experiments are being conducted in 11 major biological areas to screen for effects over a wide range of parameters in rats and mice exposed to 60-Hz electric fields for up to 120 days. Experiments in each major area use separate animal groups and are directed by professionals with specialties in the areas under investigation.

Experimental Design

Several features of experimental design are common to all the biological experiments. All experiments use Sprague-Dawley rats and/or Swiss Webster mice. All animals received from the supplier, Hilltop Laboratories, are carefully examined before being used in experiments to assure that they are healthy. A sample population from each shipment is sacrificed at delivery for virology screening and histopathologic examination. The remaining animals are maintained in standard cages in an isolation room for 1-3 weeks, earcoded, then acclimated for 14 days in cages identical to those used in the electric field exposure systems. During exposure or sham-exposure, the environment, housing and husbandry are identical for both groups of animals except for the presence or absence of the electric field. This design reduces the risk of obtaining false positive results and increases the ability to detect subtle effects of exposure. In addition, animals maintained in standard cages ("cage controls") are used in many of the experiments to verify that the exposure cages do not produce effects that might mask subtle effects of exposure.

In most of the experiments the animals are about 60 days of age at the start of exposure. Except for several cardiovascular and behavioral experiments, the field strength used for all exposures is 100 kV/m. The exposure period is about 21 hours/day; this allows about 3 hours/day for scheduled measurements, cage cleaning, feeding and watering.

With few exceptions, experiments are double-blind: the principal investigator is unaware of which animals are exposed and which animals are sham-exposed until the measurements have been completed. Most experiments are replicated to insure that the results of a single experiment are not due to some unidentified systematic error in that experiment, or to statistical chance. Our statistical criterion for positive effects is $p < 0.05$.

The results of the biological experiments with small animals that have been completed to date are summarized below. The swine study is still in progress and results are not available at this time.

Metabolic Status and Growth

In eight separate experiments, body and organ weights were determined in juvenile (age, 26 days) and young adult (age, 56 days) male and female rats, and in young adult (age, 60 days) male and female mice that were exposed or sham-exposed to 60-Hz electric fields at 100 kV/m for 30 or 120 days. In addition, food and water consumption was measured daily during the 30- to 120-day exposure periods. In other experiments, a number of parameters related to metabolism and growth were measured in rats that had been exposed to 100 kV/m for 30 or 120 days, including: oxygen consumption and carbon dioxide production rates; metabolic rates; circulating concentrations of thyroxine, thyroid stimulating hormone and growth hormone; lipid, protein, and glycogen levels in liver; and serum concentrations of glucose, urea nitrogen and triglycerides. There were no reproducible, significant differences between exposed and sham-exposed animals in any of the parameters measured in any of the experiments. These experiments failed to confirm effects reported by other investigators: increased pituitary and adrenal weights, reduced water consumption and lower body weights in rats exposed to 15 kV/m for 30 days (*14*); transitory, enhanced growth rate in mice exposed to 25 and 50 kV/m for 6 weeks (*15*); reduced growth rate in rats exposed to 50 kV/m for 100 days (*16*).

Bone Growth and Structure

Bone growth and structure were assessed in juvenile male and female rats that were exposed to 100 kV/m for 30 days starting at 28 days of age. To differentiate any possible direct effect of exposure on bone growth from that of an indirect effect on the general growth pattern of the animals, body weights, kidney weights and liver composition (fat, glycogen, protein and moisture) also were determined. Exposure had no effect on growth rate of the tibia, morphology of lumbar vertebrae or the tibia, or on cortical bone area and marrow space in the tibia. No alterations in the general growth pattern were observed.

Reproduction, Growth and Development

A series of three replicated experiments were undertaken to determine the effects of exposure to 60-Hz electric fields at 100 kV/m on reproduction and on fetal postnatal growth and development in the rat. In the first experiment, a 6-day exposure prior to and during mating did not affect the reproductive performance of either males or females. Continued exposure of the mated females through 20 days of gestation did not affect the viability, size, or morphology of the fetuses. A similar 30-day exposure of the males and unmated females did not affect their mating performance or fertility on subsequent testing. In the second experiment, exposure of the pregnant rats was begun on day 0 of gestation and continued until the resulting offspring reached 8 days of age. There were no differences between exposed and sham-exposed groups in litter sizes or survival of offspring. Body weights and subsequent growth through 42 days of age were the same for both groups. The only differences observed between exposed and sham-exposed animals were in tests of neuromuscular and neurological development through 42 days of age. A higher percentage of exposed offspring showed motile behaviors (moving, grooming and standing) and a lower percentage exhibited righting reflexes at 14 days of age. The results of the two groups were indistinguishable upon retesting at 21 days of age. Tests of the reproductive integrity of the offspring have not disclosed any deficits. The third experiment involved a 30-day exposure, beginning at 17 days of gestation and continuing through 25 days of postnatal life. There were no significant alterations in growth or development of the offspring. In another experiment mice conceived, born and raised in a 100 kV/m, 60-Hz electric field for three successive generations were compared to identical sham-exposed groups. Exposure had no apparent effect on conception, litter size, mortality or body weights up to 10 weeks of age in any of the three generations. These findings fail to confirm a report by Marino et al that exposure to electric fields of three generations of mice increases mortality and rate of growth of offspring.

Hematology and Serum Chemistry

Clinical pathologic evaluations were made in female rats exposed to 60-Hz electric fields at 100 kV/m for 15, 30, 60 and 120 days, and in female mice exposed at 100 kV/m for 30, 60 and 120 days. All experiments were replicated and used exposed, sham-exposed and cage-control animals. Hematology parameters measured included volume of packed red cells, red cell count, mean corpuscular volume, hemoglobin concentration, white cell count, distribution of leukocyte cell types, reticulocyte concentration, platelet concentration, bone marrow cellularity, red cell osmotic fragility, prothrombin time and iron uptake from plasma. Serum chemistry parameters measured included urea nitrogen, alkaline phosphatase, glutamic oxaloacetic transaminase, glucose, triglycerides, iron, total iron-binding capacity, total proteins, and protein fractions as determined by electrophoresis. Exposure of rats and mice to 100 kV/m for 15-120 days did not cause reproducible changes in any of the hematologic or serum chemistry parameters assayed. Several investigators have reported effects of electric field exposure on hematologic and serum chemical parameters (*14-16, 18-21*). The failure of this study to substantiate these reported effects may be explained by one or more of the following: 1) our exposure systems and conditions are very well defined, and eliminate secondary influences of the electric field, such as spark discharges, ozone, and noise; 2) our exposures have been replicated for each time period, whereas most other studies reporting effects have not been replicated; 3) our sham-exposed animals are more valid controls than "cage-controls" used in some studies; and 4) our electric field strengths and durations of exposure were, in many cases, different from those used by other investigators.

Immunology

A series of experiments were conducted to quantitatively assess humoral and cellular components of the immune system. Serum immunoglobulin levels were measured in rats exposed to 100 kV/m for 15 or 30 days and in mice exposed to 100 kV/m for 30 or 80 days. Complement activity and peripheral blood T- and B-lymphocytes were measured in mice exposed to 100 kV/m for 30 and 60 days. No significant differences were observed between exposed and sham-exposed rats or mice in any of the parameters measured. In the second phase of this study, we assessed functional immune responses by measuring serum antibody levels in response to a specific antigen and by in vivo assays of cell-mediated immunity. To assess the primary immune response, the serum antibody levels were measured in mice exposed to 100 kV/m for 30 or 60 days and challenged with keyhole limpet hemocyanin (KLH). No significant differences in the amount of precipitating antibody for KLH were observed between exposed, sham-exposed and cage-control mice. Cell-mediated immunity was evaluated in mice after 30 days of exposure to 100 kV/m by determining the delayed hypersensitivity to KLH and the contact sensitivity to dinitrofluorobenzene (DNFB). Exposure had no effect on the response of sensitized mice to DNFB. In exposed mice, however, there was a significantly reduced response to KLH as measured by skin thickness at the site of injection. The mean reaction diameters for the two groups were not significantly different. Other experiments have been made in which cell-mediated immunity was tested in mice exposed for 30 to 180 days by examining the response of lymphocytes stimulated with mitogens. No effects were observed.

Endocrinology

Plasma hormone concentrations, and endocrine gland and reproductive organ weights were examined in rats after exposure to a 60-Hz electric field at 100 kV/m for 30 days. In addition, a detailed examination was made of male reproductive endocrinology. This included plasma gonadotrophin levels, plasma testosterone, testicular vein androgens, blood flow rate through the reproductive tract and androgen secretion rates. Exposure of rats to electric fields had no effect on body weight, weights of endocrine organs or plasma levels of corticosterone, thyroxine, thyroid stimulating hormone, follicle stimulating hormone, testosterone or luteinizing hormone. Testicular weights were unaffected by the treatment, and there was no effect on testicular blood flow rates, androgen levels, or testosterone secretion rate. These data are in conflict with previous studies (*14,20*) where much lower field strengths were employed, and suggest that secondary factors (electrical discharges, corona and ozone) may have had a major influence on those experiments. Our results are in general agreement with those of the Italian (ENEL) studies summarized by Cerretelli and Malaguti (*22*), where field strengths of 80-100 kV/m were used.

Cardiovascular Function

A series of experiments were conducted to determine whether exposure to 60-Hz electric fields alters cardiovascular function in the rat. Parameters assessed included electrocardiograms (ECG), heart rate, blood pressure and vascular reactivity. In addition, the cardiovascular and endocrine responses of rats to acute cold stress were determined in animals that had been exposed to 60-Hz electric fields. Electrocardiograms and heart rates were evaluated in male rats exposed to 80 kV/m for 8 hours or 40 hours (8 hours/day for 5 consecutive days), and to 100 kV/m for 1 month (~ 21 hours/day). The same evaluations were made on female rats exposed to 100 kV/m for 1 or 4 months (21 hours/day). ECG analysis consisted of measuring the durations of the PR interval and the QRS complex. Recordings were made during the first hour after the completion of electric field exposure. No differences were seen between exposed and sham-exposed animals in any of the experiments. These results failed to confirm the findings of Blanchi et al (*23*), who reported that exposure of rats to 100 kV/m slowed electrical conduction in the heart. Systolic, diastolic, pulse and mean systemic blood pressures, and vascular reactivity were measured in rats that had been exposed to 100 kV/m for 35 or 120 days. Vascular reactivity was quantified by measuring the change in pulse pressure in response to an injected dose of phenylephrine, a vasoconstrictor that produces an elevated pulse pressure by increasing peripheral resistance to blood flow. No significant differences were observed between exposed and sham-exposed animals in any of the measures. Cardiovascular and endocrine responses to cold stress were measured in rats that had been exposed or sham-exposed to 100 kV/m for 30 days. Plasma corticosterone and thyroid stimulating hormone levels were determined in the animals prior to and after being subjected to acute cold stress (-13 ± 1°C) for 1 hour. No significant differences were found between exposed and sham-exposed rats before or after cold stress. In a parallel experiment heart rate, skin temperature and deep colonic temperature were measured during cold exposure in rats that had been exposed or sham-exposed to 100 kV/m for 30 days. Values of both groups were essentially identical.

Pathology

Complete gross and histopathologic examination of approximately 30 selected tissues per animal were performed on groups of male rats and both male and female mice exposed or sham-exposed to a 100 kV/m, 60-Hz electric field for 30 or 120 days. Animals were weighed after sacrifice, and selected organs from each animal were weighed. No histopathologic effects of exposure were observed, and exposure had no significant effect on body or organ weights. The results of these studies fail to confirm a report by Soviet scientists (*19*) that exposure of rats to electric fields at 5 kV/m produces vascular damage and atrophic changes in brain and liver.

Central Nervous System

A detailed histologic examination was made of the brains of mice exposed to 100 kV/m for 30 days. Results failed to reveal any differences between exposed and sham-exposed mice.

Neurophysiology

Synaptic transmission and peripheral nerve function were assessed in male rats that had been exposed to 100 kV/m for 30 days. Immediately following exposure or sham-exposure, superior cervical sympathetic ganglia, and vagus and sciatic nerves were removed from rats anesthetized with urethan, placed in a temperature-controlled chamber (37.5 ± 0.4°C) and continuously superfused with a modified mammalian Ringer's solution equilibrated with 95% O_2 and 5% CO_2. Parameters used to characterize synaptic transmission and peripheral nerve function were: 1) characteristics of the postsynaptic or whole-nerve compound action potential (including amplitude, area, configuration, rate of rise, and rate of fall); 2) conduction velocity of the postsynaptic or whole-nerve compound action potential, measured at the beginning of the compound action potential (inflection conduction velocity) and at the peak of the compound action potential (peak conduction velocity); 3) absolute and relative refractory periods; 4) accommodation; 4) stimulus strength-duration relationships; 6) changes in poststimulus excitability (e.g., conditioning-test response); 7) frequency response; 8) post-tetanic response; 9) high-frequency-induced fatigue. The only consistent difference observed between exposed and sham-exposed animals was a shift in the conditioning-test response curve for exposed animals that suggested increased synaptic excitability.

Behavior

A series of experiments were conducted to assess the effects of various levels of 60-Hz electric fields on the behavior of rats. These experiments have focused on perception and passive avoidance behavior, activity during exposure and performance of a learning task immediately after exposure. In the passive avoidance experiments, rats were given the option of being exposed to or shielded from various field strengths during short- (45-minute) and longer-duration (∿ 24-hour) tests. Rats were placed in a shuttlebox, one end of which was shielded and the other end visually identical but unshielded from the 60-Hz electric field, and scored for activity (number of end-to-end traverses) and time spent in each end of the shuttlebox. In short-duration tests (45 minutes), rats exposed to field strengths $\geq$ 90 kV/m spent significantly more time on the shielded side of the shuttlebox than did the sham-exposed controls. Repeated tests over 4 weeks, one test per week, did not modify this behavioral response. However, exposure of the rats to 100 kV/m for 30 days (630 hours) prior to the test, attenuated this behavioral response. Activity during the 45-minute tests was significantly greater in exposed rats than in sham-exposed controls. This higher activity was seen at field strengths as low as 50 kV/m and increased with repeated tests. In longer-duration tests ($\sim$ 24 hours, 12 hours light:12 hours dark), rats exposed at 75 or 100 kV/m spent more time in the shielded end of the shuttlebox than did controls during both the light and dark periods. However, rats exposed at 50 kV/m spent more time in the field than controls, but only during the 12-hour light period. The results of the longer-duration tests confirmed the results of the 45-minute tests in which exposed rats showed an increased activity. This effect was observed in rats exposed to a field as low as 25 kV/m and occurred only during the first hour of exposure. An experiment was conducted to assess the performance of rats in a learning task after exposure to 60-Hz electric fields at 0, 50 or 100 kV/m for 23.5 hours. After exposure, each rat was placed in a two-compartment shuttlebox and learned to move from one compartment to the other in response to an 80-dB tone in order to avoid a 20-second shock to the feet (an avoidance response). If no avoidance response was made by the rat, it could escape the ongoing shock by moving to the other compartment (escape response). No consistent differences were observed between exposed and sham-exposed rats in avoidance or escape responses.

SUMMARY

Exposure of rats and mice to 60-Hz electric fields at 100 kV/m for up to 120 days had no statistically significant, reproducible effects on a number of measures of metabolic status and growth, bone growth and structure, reproduction, hematology and serum chemistry, endocrinology, cardiovascular function, nerve function, or organ and tissue morphology. An effect on cell-mediated immunity was detected and is being evaluated further in additional experiments. Exposure of rats in utero (day 0 of gestation to 8 days of age) had a transient effect at 14 days of age on motile behavior and development of the righting reflex. Significant effects were observed in synaptic transmission and behavior. Exposure to 60-Hz electric fields may increase the excitability of the nervous system of rats. Experiments are in progress to obtain a better understanding of these effects and their potential consequences.

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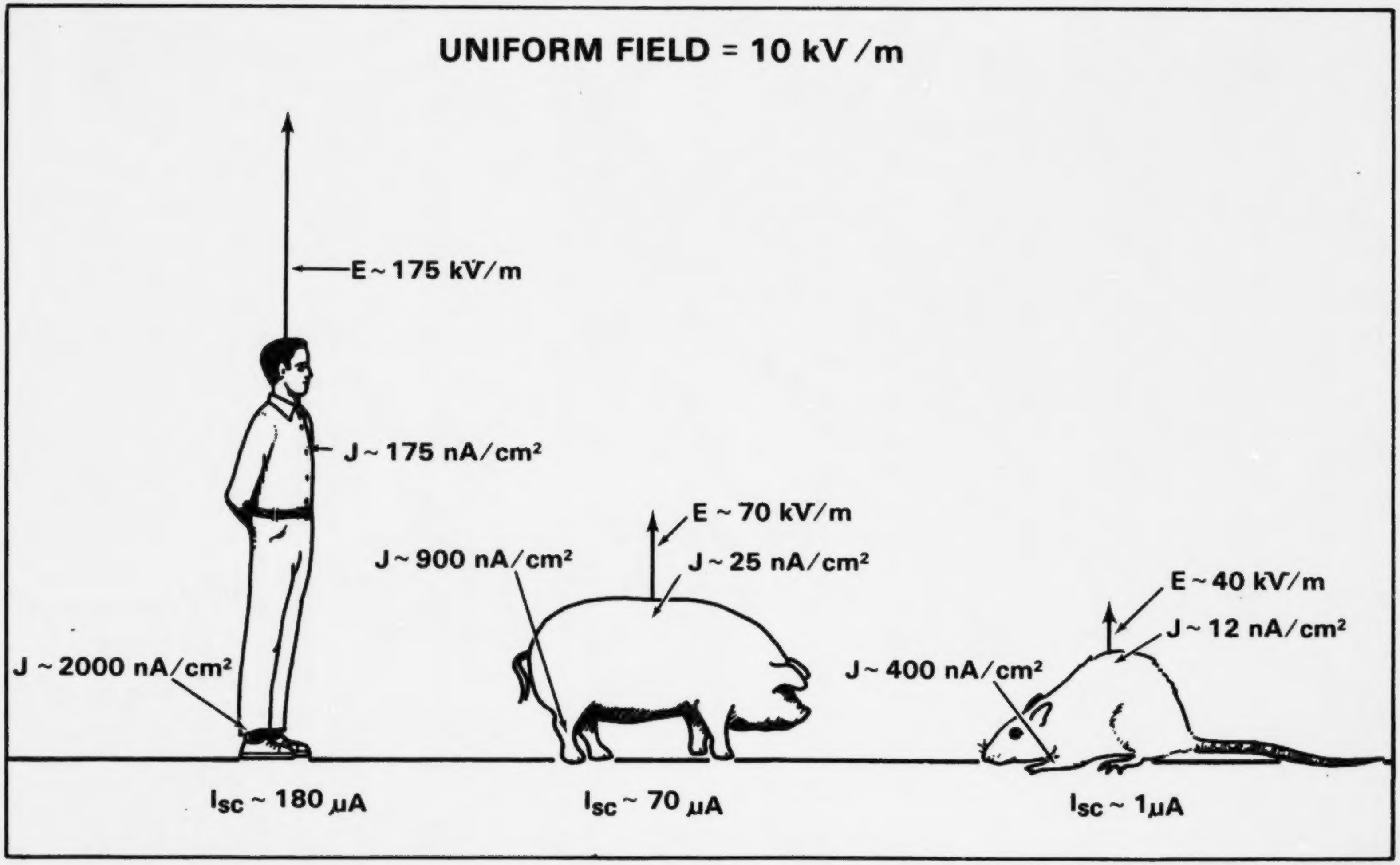

Figure 1. Interaction of man, pig and rat with a uniform, 10 kV/m electric field. E represents the enhanced field strength at the highest point on the surface of the subject; J is the cross-sectional current density at the trunks and lower limbs of the subjects; I_{sc} is the short-circuit current between the subject and ground.

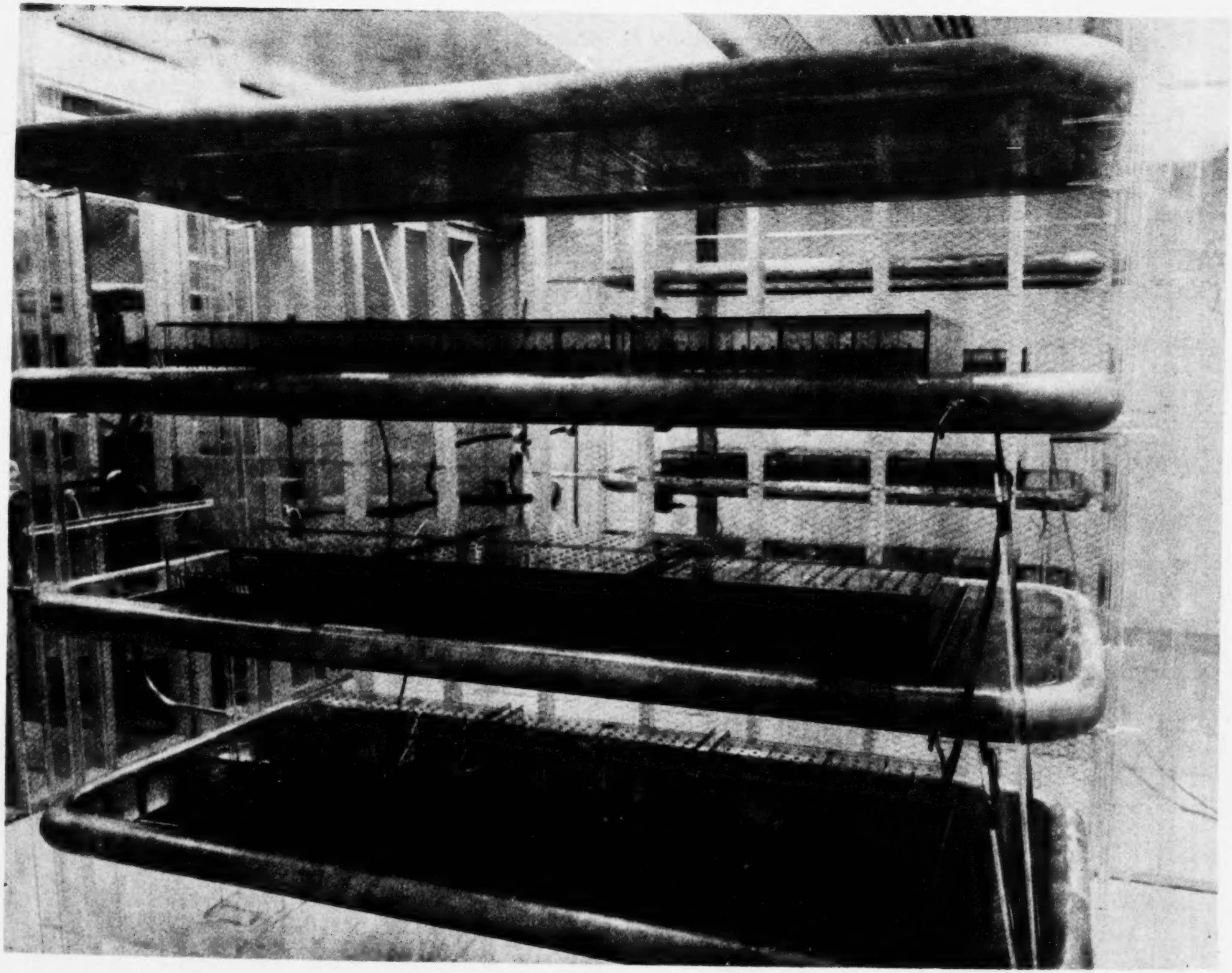

Figure 2. System used to expose rats to 60-Hz electric fields.

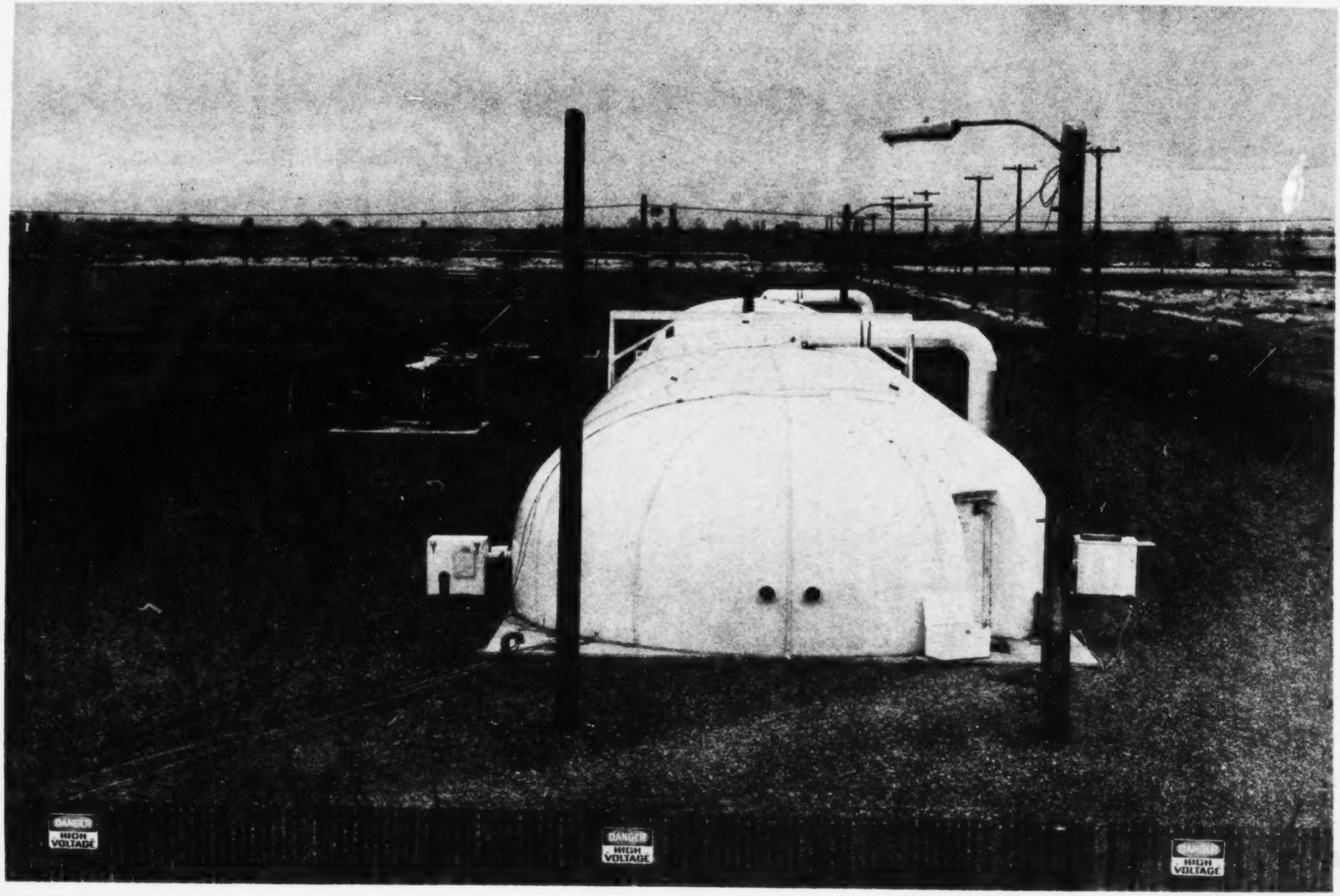

Figure 3. Exterior view of buildings used to house swine during exposure or sham-exposure to 60-Hz electric fields.

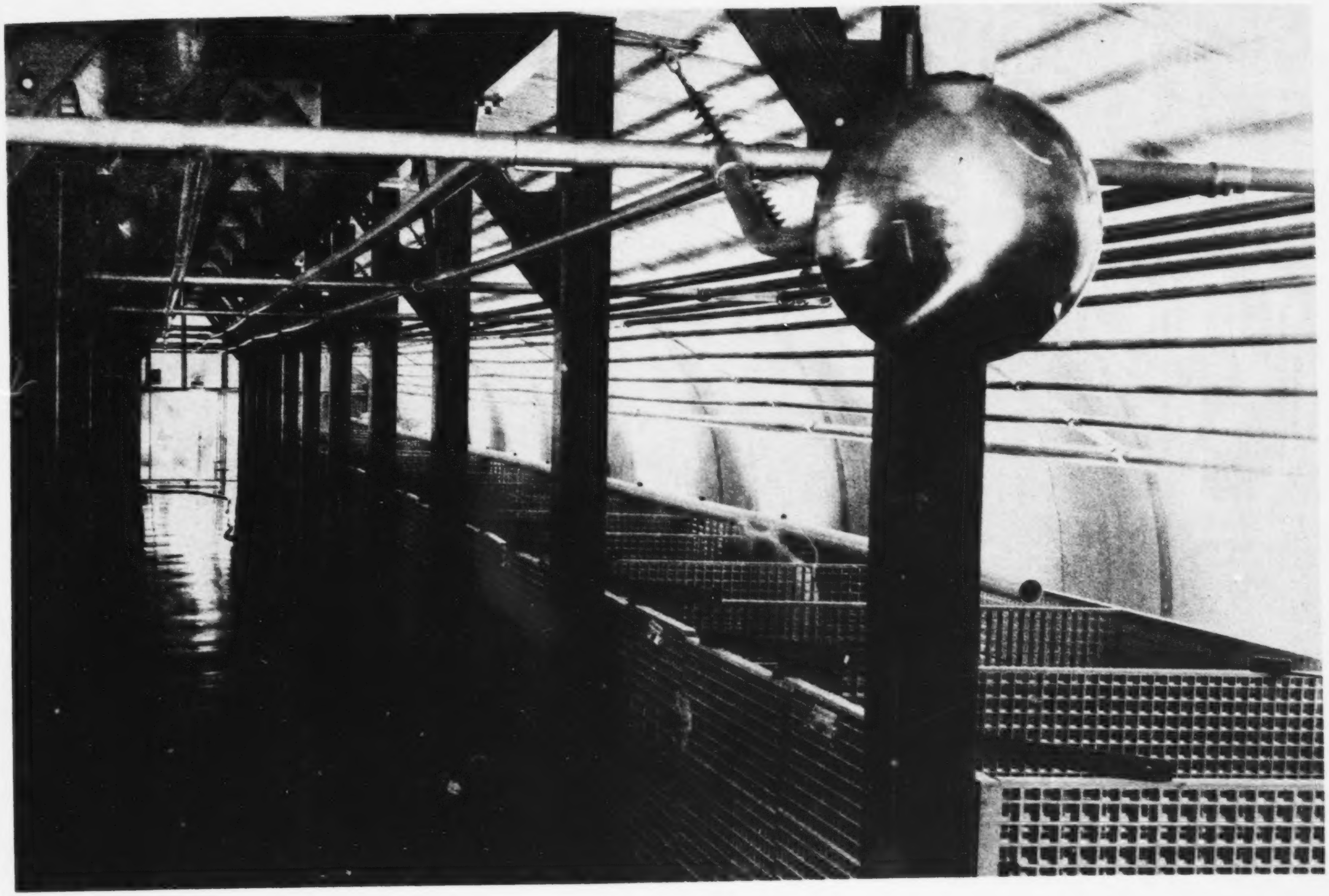

Figure 4. Interior view of exposure building showing overhead electrodes and stalls for individual swine.

HEALTH BENEFITS AND RISKS OF ELECTRIC POWER CONSUMPTION

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*The contents of this paper are the responsibility of the author and should not be attributed to EPRI.

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So far we have concerned ourselves with a traditional environmental view of electricity generation: the health and environmental costs of fuel extraction and transport, electricity generation with its accompanying air pollution, solid waste disposal, and electricity transmission. Tomorrow we will discuss those environmental concerns peculiar to nuclear power generation. As we discuss these issues, I can't help but think that somehow we're missing a fundamental aspect in the current environmental debate, that of how much energy we should consume. Environmental regulations are seen as tools to help limit the consumption of energy, even though they were created for other purposes.

There is no doubt that environmental concerns are justified. The serious air pollution episodes of Donora, London, the Meuse Valley have had environmental consequences that should never be repeated, and we now have laws to insure against a reoccurrence. SO_2 and particulate levels have declined considerably in our cities as air pollution control measures have been taken. We spend much time debating over appropriate levels of SO_2 and whether sulfates are injurious to health, but new and existing regulations are observed. Certainly there is a need to review these and all regulations periodically as required by the Clean Air Act. It is encouraging that the 24-hour ambient standard for photochemical oxidant was recently relaxed. The fact that standards can be relaxed as well as tightened may suggest that we are approaching socially optimum standards rather than seeking an unrealistic environmental perfection.

Despite the original importance of health issues in developing environmental standards, the most recent controversies over proposed power plants avoid debates about health effects. This is certainly true for the substances most commonly emitted by fossil fuel power plants. Current issues of concern are visibility degradation, acid rain, socio-economic impacts, aesthetic impacts and impacts upon non-human species. It is noteworthy that the Seabrook controversy did not focus upon health and safety risks of nuclear power, but upon the fate of a clam population. Health effects of air pollution were all but ignored in the Kaiparowits debate. It was to be the cleanest coal-burning power plant ever built. Health effects were not an issue; concern for visibility, aesthetics and water quality were most often voiced.

The Kaiparowits debate does in fact provide an important clue to what may be a new and important environmental issue. The Environmental Protection Agency (EPA), in its critique of the Environmental Impact Statement (EIS), focused attention on the need for Kaiparowits (1). The EIS, according to the EPA, did not consider the impacts of conservation measures being implemented by the State of California. These included mandatory insulation standards, a partial ban on electric heating in new commercial buildings, appliance efficiency standards, and commercial lighting standards. Moreover, the EPA was concerned that the EIS failed to address the possible demand-reducing effects of peak-load pricing and other load-management techniques. Environmentalist and other groups agreed. Friends of the Earth criticized the methods used to forecast electricity demand and argued that "financial incentives and disincentives combined with regulations concerning electrical power use could limit electric power demand to the point where new generation capacity would not be required" (2). The California Governor's Office of Planning and Research asked "if demand forecasts overestimate the actual electricity need and a surplus of electric energy is available, growth will be encouraged to fulfill the demand forecasts and proposed conservation efforts will be curtailed" (3). In other words, growth should be curtailed in order to encourage conservation.

It is my argument that one of the most important current objectives of environmental concerns is the desire to impose a conservation program upon society. The origin of this attitude may be environmental, as one way to preserve our environment is to allow no new intrusions and to redistribute existing resources to meet current needs. What better way to achieve this goal than to control energy growth.

NEPA or the National Environmental Policy Act and other regulations force us to scrutinize every action or development and to list all conceivable environmental impacts. If anything is scrutinized, some impacts will be found. Such scrutiny is useful to identify potentially important impacts, but it can also divert our attention from the fundamental issues which underlie societal concerns. Such I believe is the case of Seabrook; very few members of the Clamshell Alliance worry about the future population of the seaclam; nuclear power and level of energy consumption are the unspoken issues.

I don't argue here that NEPA should be abolished or amended, but while attending to the risks of electricity generation, we must remember the total picture. It is important to foresee impacts on clam populations, but we must at the same time ensure that we are providing enough energy for our society. Environmental regulations are not the appropriate tool to achieve the universally-accepted goal of energy conservation.

There are two ways in which environmental policy can lead to reduced electricity consumption: (1) it can impose environmental constraints which increase the costs of electricity and therefore reduce demand; (2) it can use environmental regulations to restrict ~~the~~ the development of new energy facilities and thereby reduce the supply.

Electricity costs can be increased in many different ways through environmental regulation: air and water control equipment, higher-priced fuels, environmental siting and permitting procedures, delays in construction and operation. All of these costs must be weighed against the environmental benefits they yield, but we must also weigh these benefits against the social consequences of the higher costs. Higher electricity costs contribute to inflation, that problem which troubles the U.S. public most according to the several public opinion polls reported in our newspapers. Inflation itself spawns many socio-economic impacts, also cited frequently these days in our newspapers. Some attention is presently being paid to this issue. It has been raised by the Council of Economic Advisers, dismissed by the EPA, and is now under study by a commission responsible to Alfred Kahn, our national inflation fighter.

It may be difficult to weigh environmental concerns against inflationary concerns, particularly when there is uncertainty and debate about both. The two are certainly intertwined; better estimates of their association are needed so that our policy decisions can reflect them judiciously.

Price increases of electricity can achieve conservation, but responses are limited, particularly in the short-run where voluntary restraint or changes in lifestyle are the only significant options. There is little opportunity to introduce technical innovations through equipment modification, alternative production cycles and technical systems in the short-run. Ten years as a minimum are required for these innovations to come to pass. Other responses such as fuel substitution or changes in the industrial mix take even longer to implement.

As an energy conservation tool, price increases induced by environmental regulations will impact the residential sector the greatest. Little immediate conservation will take place in the industrial and transportation sectors because the potential for conservation in these sectors resides largely in technical innovation (which requires a lead time of at least five years). There may be some possibility for conservation in the commercial sector, but it is the residential sector who will respond the most, and since taxes on energy are regressive, it is the lower income groups who will be the most affected by increases in energy prices.

Figure 1 demonstrates the increase in fuel costs resulting from environmental regulation according to Consolidated Edison of New York (4). These costs represent the difference in cost between high and low sulfur fuel. Similar cost differentials exist for other fuel uses; i.e., residential and commercial boilers. Environmental benefits may have been achieved as a result of these costs; certainly SO_2 levels have been reduced significantly in New York City over this period. (See Figure 2)(5). There has incidentally been no concommitant measurable change in the health status of New York City during this period (6).

Dr. Lawrence E. Hinkle, Director of the Division of Human Ecology at the New York Hospital-Cornell Medical Center worries about the health effects of increased energy costs.

He writes that "in the past few years, as the cost of home heating and electricity have gone up, an increasing number of landlords of the poor have reduced or abolished the heating of dwellings and have turned off the electric power of those who could not pay their bills. In New York we actually have poor old people dying from cold and exposure in unheated apartments, and we have people of all ages dying from accidental fires, smoke inhalation, and carbon monoxide poisoning because they have attempted to supplement the inadequate heat in their apartments by turning on the oven or by using a charcoal brazier or a kerosene space heater." Dr. Hinkle adds, "It seems quite possible that further efforts to lower the SO_2 content of the outdoor air to produce health benefits that are probably illusory may in fact increase morbidity and mortality from smoke fires, and carbon monoxide"(7).

Writing in the Annals of Internal Medicine, Dr. John E. Milner, Department of Environmental Health, University of Washington, reports of several British studies which demonstrate a high incidence of hypothermia among the elderly poor, who cannot afford to heat their homes to adequate temperatures (8). One British study in fact suggested that between "20,000 and 100,000 hypothermia deaths occurred yearly in Britain". Hypothermia is the failure to maintain a body temperature adequate for optimal physiologic functioning, and it can result in depressed respiration and consequent pneumonia, pancreatitis, heart failure, and cerebrovascular accidents. There is no information about the incidence of hypothermia in the U.S., but the British experience should cause some concern about the potential for hypothermia in the U.S., particularly in an era of rapidly-rising energy prices.

I or no one else knows for certain how many deaths or health effects can be attributed to hypothermia or unsafe heating resulting from high energy costs. We have ignored these impacts to date and have certainly not considered their possibility in our framing of regulations. Everything is more complicated than previously thought to be; environmental policy-setting is no exception. These policies can have negative impacts as well as benefits.

Environmental regulations may yield some conservation results as a side-effect, but they are not designed for that purpose and are not the most suitable tools for achieving conservation; their results may in fact be counterproductive, yielding more societal harm and discomfort than intended. The tools for implementing conservation need to be designed and evaluated for that purpose alone.

Environmental regulations can yield some conservation by limiting supply as well as limiting demand. Availability of supply was an issue in the Kaiparowits debate. The cancellation of a power plant can lead to environmental benefits and possibly to some conservation. But as a conservation measure, the most significant response possible in the short-run to the non-building of power plants is the voluntary change in lifestyle which will take place largely in the residential sector. Some of the drawbacks associated with this approach were cited above; others are a result of a general shortfall in electricity generating capacity.

The amount of conservation possible is limited. A detailed study of the potential for technological conservation suggested that a seventeen percent reduction in anticipated energy use could reasonably be attained through conservation measures by 2000 (9). (See Table 1.) With extreme efforts this reduction could be as high as 34%. Demographics favor an increase in total energy consumption. According to the Bureau of Labor Statistics the total number of Americans expected to be in the labor force in 2000 A.D. is 119 million contrasted with the 102 million in the labor force today. Past increases in productivity have generally occurred as a result of replacing manpower by machine power which requires a greater per capita energy consumption. It is increased productivity which generally contributes to our increased per capita incomes. The larger share of elderly in our 2000 A.D. population will also lead to greater energy requirements.

Environmental requirements in themselves will demand additional energy. For example, a change from water-cooled to air-cooled condensors, in order to reduce the detrimental effects upon water resources would require increased fuel consumption of 6-10% for fossil fuel plants and 7-13% for nuclear power plants. Flue gas desulphurization will require about 5-10%

of the input heat of a fossil fuel power plant.

In addition the extraction of energy fuels will require more energy as they become less readily available and as environmental measures are needed to restore the land. All of these factors lead us to estimate that by the year 2000, we would need significant additional energy to maintain our standard of living. Our current consumption is about 75 quads a year. One can argue about forecasts of the energy demand for the year 2000, but no one argues that our energy supply must increase between now and then if we are to improve or even maintain our standard of living. Even Lovins estimates that we will consume 95 quads of energy in the year 2000, although Lovins would suggest that very little of this energy would be in the form of electricity (10). Given that the year 2000 is but 21 years away, and that the existing trend is towards electrification, a significant share of the energy growth will be in the electricity sector.

Insufficient electricity supply can create severe problems. We noted above some of the problems which could occur in the residential sector. In addition, decreased capacity will provide a smaller reserve margin and reduced reliability of service. Certainly the probability of blackouts and brownouts would increase. An improbable combination of natural phenomena, improperly operating protective devices, and communication difficulties all led to the New York City blackout of July 13, 1977. Constraints on electricity supply will certainly increase the probability of such events. The events of the catastrophic New York City blackout are well-known. In a study for the Department of Energy (11), Systems Control, Inc. estimated a lower bound of $345 millions damage. (Note Table 2.) The greatest losses were those which received little publicity: loss in business activity and losses to the utility, Con Ed. Vandalism, crime, and food spoilage contributed only a small part of the total damage.

There may have been significant health effects, also. Table 3 gives mortality by cause of death for several days around the blackout. We note that the blackout itself began at 9:36 p.m. on July 13 and ended in most areas the evening of July 14. The most striking statistic is the increase in cardiovascular-renal deaths among those 65 years or older during the day after the blackout began. With only one data point, no adjustment for weather, and no consideration of the usual variance in the New York City daily mortality rate, it is dangerous to draw any conclusions. Nevertheless the figures are consistent with a hypothesis that blackouts can lead to increased mortality.

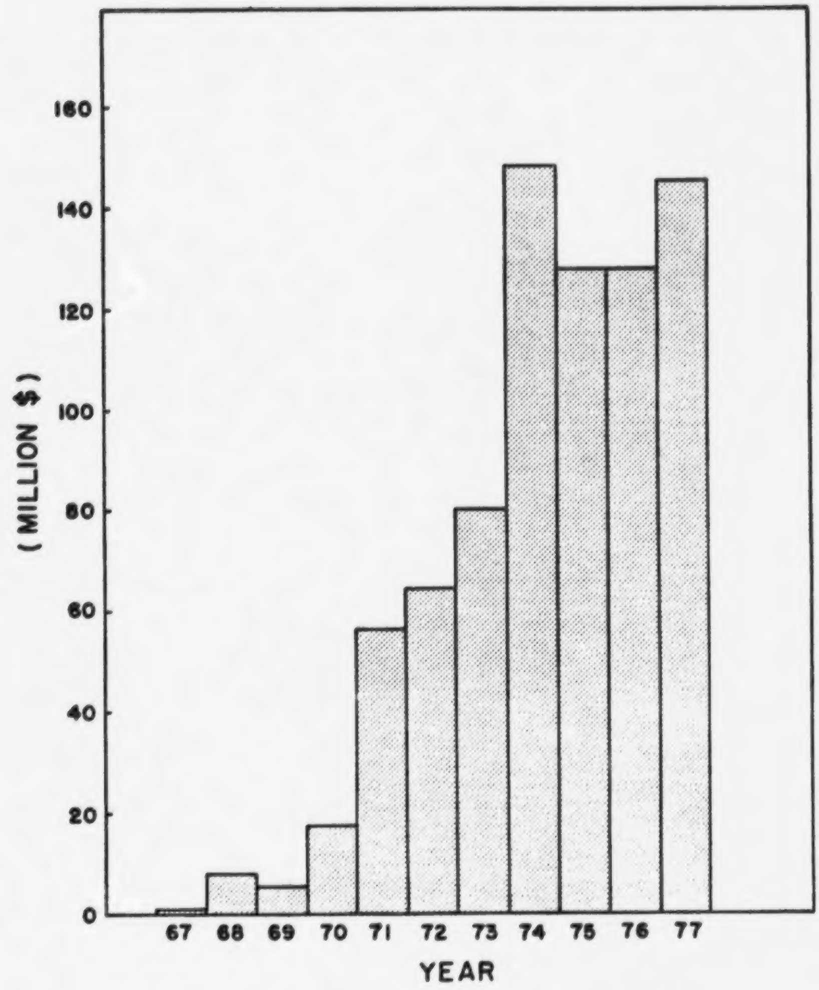
The damage associated with the New York City blackout was not unique; cost estimates of other power outages have been made and are given in Table 4. The higher costs of the 1977 New York City outage may reflect its long duration, but all of the estimates suggest that blackouts can be costly.

The other danger associated with a shortfall in capacity is that inadequate supply will stunt economic activity. This can happen in two ways: In periods of inadequate supply, the industrial sector is often the first to whom supply is curtailed. This was the case with the recent natural gas shortage. In the face of an uncertain supply, no industry will locate in a given area. Secondly, frequent curtailments or outages can create a generally unfavorable economic climate with little confidence for investors. In both cases, limited or lessened investment will have its effects on the economy. It will result in higher unemployment which is far less benign than the monthly figures often bandied about. A study undertaken by Dr. Harvey Brenner, School of Hygiene and Public Health, The John Hopkins University, relates national health statistics to unemployment(12). The results of his study are summarized in Table 5. We see that general mortality, infant mortality, cardiovascular mortality, cirrhosis mortality, suicide rates, homicide rates, imprisonment rates, and mental hospital admissions rates are all statistically significantly and positively associated with higher unemployment. General mortality, infant mortality, suicide rate, imprisonment rate, and mental hospital admission rates were all positively and statistically-significantly associated with inflation rates. Brenner replicated his results for three states (California, Massachusetts, and New York) and for two countries (England and Wales, and Sweden). There is a need to scrutinize Brenner's data and methodology, but his results suggest that we pay close attention to how we impact our economy.

My underlying concern in this paper is that we pay full attention to both the risks and benefits of electricity consumption. At present we highlight the environmental risks associated with electricity generation and we applaud the generation of less electricity in the name of conservation. Conservation is a worthy goal, but reduced electricity use is not equivalent to conservation. Measures aimed merely at restricting energy use may have public health consequences.

Environmental policy is naturally directed towards protecting all site-specific and regional environments. When it is applied in the energy sector, it is applied on an incremental plant-by-plant basis. The results are that the broader policy issues of adequate energy supply are not addressed. We desperately need an energy policy with an important conservation component so that our society and its regulations can address energy concerns along with environmental concerns in order to develop solutions which minimize the total social impact.

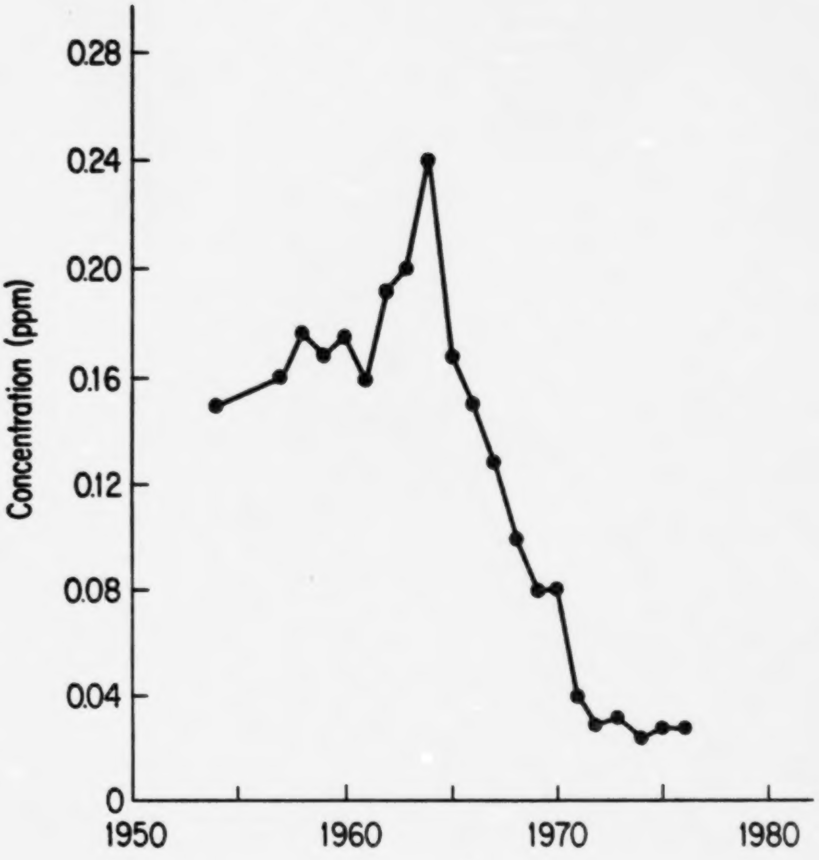
Figure 1



ANNUAL COST OF SULFUR DIOXIDE CONTROL IN POWER AND STEAM PLANTS LOCATED IN NEW YORK CITY

Source: Reference (4).

Figure 2



CITYWIDE ANNUAL AVERAGE SO_2 CONCENTRATION (1954-1976)

Source: Reference (5)

Table 1

YEAR 2000 END USE CONSERVATION POTENTIAL PERCENT SAVINGS

(IN PERCENT)

CONSERVATION PROGRAM	ELECTRIC ENERGY SECTOR	NONELECTRIC ENERGY SECTOR	TOTAL ENERGY
NONE	0	0	0
REASONABLE	17	25	20
EXTREME	34	50	40

Source: Reference (9).

Table 2: SUMMARY OF ECONOMIC IMPACTS[1]

Impact Areas	Direct ($M)		Indirect ($M)	
Businesses	Food Spoilage	$ 1.0	Small Businesses	$155.4
	Wages Lost	5.0	Emergency Aid (private sector)	5.0
	Securities Industry	15.0		
	Banking Industry	13.0		
Government (Non-public services)			Federal Assistance Programs	11.5
			New York State Assistance Program	1.0
Consolidated Edison	Restoration Costs	10.0	New Capital Equipment (program and installation)	65.0
	Overtime Payments	2.0		
Insurance[2]			Federal Crime Insurance	3.5
			Fire Insurance	19.5
			Private Property Insurance	10.5
Public Health Services			Public Hospitals--overtime, emergency room charges	1.5
Other Public Services	Metropolitan Transportation Authority (MTA) Revenue Losses	2.6	MTA Vandalism	.2
	MTA Overtime and Unearned Wages	6.5	MTA New Capital Equipment Required	11.0
			Red Cross	.01
			Fire Department overtime and damaged equipment	.5
			Police Department overtime	4.4
			State Courts overtime	.05
			Prosecution and Correction	1.1
Westchester County	Food Spoilage	0.25[3]		
	Public Services equipment damage, overtime payments	0.19		
TOTALS		$55.54		$290.16

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[1] Estimate based on aggregate data collected as of May 1, 1978. See previous page for discussion of limitation of these costs.

[2] Overlap with business losses might occur since some are recovered by insurance.

[3] Looting was included in this estimate but reported to be minimal.

These data are derivative, and are neither comprehensive nor definitive.

Source: Reference (10).

Table 3

Cause of Death July 12-15, 1977

		Total				Respiratory			Cardiovascular Renal			Homicide			Other		
Date	July	12	13	14	15	13	14	15	13	14	15	13	14	15	13	14	15
Age	64-	--	72	77	68	6	11	8	24	25	20				39	37	36
	65+	--	104	157	139	14	15	12	51	100	87				38	42	39
Total		212	176	234	207	20	28	20	75	125	107	4	6	5	77	79	75

Blackout began 9:36 p.m. July 13, 1973; power restored c. 10:30 p.m., July 14, 1977

Source: Reference (11).

Table 4

COMPARATIVE COST ESTIMATES OF POWER OUTAGES

	Geographic Scope	Estimated Cost	Date
Hauugard[1]	New York State	\$2.17 million/hr	1971
NYEDA[1]	NYC	\$2.5 million/hr	1971
Shipley[1]	U.S.	\$.60/kwh	1971
Telson[1]	New York State	\$.33/kwh	1973
NERA[1]	U.S.	\$1/kwh	1976
Gannon[3]	U.S.	\$2.68/kwh(ind), \$7.21/kwh(comm)	1976
Ontario Hydro[2]	Canada	\$15/kwh(15min), \$91/kw(1 hr)	1977
Present Study[4]	New York City, 1977	\$4.11/kwh	1978

[1] As reported in Myers

[2] Ontario Hydro, "Ontario Hydro Survey on Power System Reliability: Viewpoint of Large Users", April, 1977.

[3] P. E. Gannon, "Cost of Interruptions: Economic Evaluation of Reliability", May, 1976.

[4] Direct plus indirect costs divided by estimated kwh sales lost due to the blackout (see Section 4). Disaggregated, direct was \$.66/kwh and indirect was \$3.45/kwh.

Source: Reference (11).

Table 5: MULTIPLE REGRESSION OF NATIONAL ECONOMIC INDICES ON SELECTED MORTALITY RATES, UNITED STATES

(t--Statistics in parentheses)

Dependent Variable	Years	Intercept	Time Trend	Log Time Trends	Per Capita Income	Unemployment rate	Inflation Rate	R^2	F	D.W.
GENERAL MORTALITY RATE[1] (Lag 0-5)[3]										
(1) Mortality rate, total whites	1940-74	96.2			-0.53E-2 *(1.92)	0.62 *(5.05)	0.87 *(2.96)	0.89	*21.9	1.93
(2) Mortality rate, total nonwhites	1940-74	136.4			-.32E-1 *(6.55)	1.68 *(6.95)	2.83 *(5.16)	.96	*74.1	1.67
(3) Mortality rate, LT 1 whites	1940-74	497.7			- .24 *(7.06)	12.65 *(8.01)	3.59 *(5.44)	.97	*98.6	2.11
(4) Mortality rate, LT 1 nonwhites	1940-74	936.2			- .43 *(10.48)	17.74 *(10.69)	30.77 *(6.49)	.99	*199.6	2.40
(5) Cardiovascular disease mortality rate[2] (Lag 1-4)[4]	1940-73	-2,836.8	-17.8 *(4.70)	1,077.0 *(4.76)		2.85 *(1.83)	1.04 (.78)	.74	*6.50	2.36
(6) Cirrhosis mortality rate[2] (Lag 0-5)[3]	1940-73	-.2			.65E-2 *(8.99)	.14 *(4.21)	.16E-2 (.01)	.98	*114.4	1.65
(7) Suicide rate[1] (Lag 0-5)[3]	1940-73	6.34			.90E-3 †(1.50)	.42 *(14.23)	.27 *(4.23)	.91	*26.2	1.80
(8) Imprisonment rate[1] (minus 1942-45) (Lag 0-2)[4]	1935-65	-577.90	-3.18 *(3.14)	195.5 *(3.48)		1.59 *(5.92)	.64 *3.35)	.76	*10.4	1.87
(9) Mental hospital admission rate LT 65[1] (Lag 0-5)[3]	1940-71	-70.00			.49E-1 *(8.69)	3.11 *(B.92)	2.19 *(3.21)	.97	*63.4	1.85

---

1 Per 10,000 population
2 Per 100,000 population
3 2d degree, polynomial distributed lag equation.
4 Ordinary least squares equation.

1 - tailed tests:

†significant at 90 percent level of confidence: t=31.1; F=1.89

*significant at 95 percent or greater levels of confidence, i.e.: at 95 percent level of confidence.

t=1.71; F=2.28; at 99 percent level of confidence, t=2.49; F=3.31; at 99.9 percent level of confidence, t=3.45; F=4.71.

Source: Reference (12).

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DISCUSSION

SESSION IV

John Blair, Evansville, Indiana: Most of my remarks are going to be addressed to Dr. Wyzga from the Electric Power Institute, and if he would like to respond, I would appreciate it. It seemed to me as he was talking, that I was going to go back to Evansville, Indiana, and watch the 20 or so power plants that are going up around my home with a lot different light because I was hoping that they would put out pollution so my electrical costs could stay down. It seems as though he failed to recognize one very essential element of economics, as far as electric power production is concerned, and that is, according to the New York Times yesterday, we are 36% over the peak load capacity needed in this country. Now 36% over our peak load need says that there is an inflation built in, in economic aspects of utilities in this country, that is totally unnecessary. Any time you are operating 36% over peak load capacity, that means you are paying for power plants that aren't needed. I'd like to point out two examples of this, the fact that power plant construction is what causes the price of electricity to go up. One of them is the Marble Hill Plant which is being built on the Ohio River at Madison, Indiana, at the cost of approximately two billion dollars. It is a nuclear plant. That is doubling the capital base of the company that is building it, the Public Service Company of Indiana. Doubling it! Their rates are based upon their capital base. That looks to me like it is going to double their rates. And it is not pollution control equipment that is going to cause the rates to double.

I would also like to point out the example of the Indiana-Michigan Electric Company Power Plant, which is owned by the American Electric Power, that is going up in Rockport, Indiana, which is also a 2,400 megawatt unit. It also is going to double Indiana-Michigan's capital base. That is why electric rates go up; not just becase scrubbers or precipitators or whatever, are put on these. I would also like to point out that conservation does work. As a matter of fact, conservation has taken place so much in Indiana over the last year that the construction of several power plants has been delayed. The

Indiana-Michigan Plant, for instance, has been delayed by at least a year because Indiana-Michigan misprojected their electrical needs. And also, during the coal strike in 1977-78, we found that in Indiana a 25% reduction in electrical usage was accomplished with the lay-off of 6,000 people under an emergency situation. Now 25% electrical reduction in the state of Indiana was a pretty massive reduction. Those 6,000 people who were laid off, had adequate planning taken place, probably would not have been forced to be laid off. Also another thing is that peak load demand is hardly a way to run this country whenever we are having a real serious environmental problem. Peak load says that you can build and accomplish the needs of whatever you want. Environmentalists, it seems, would say, let's go with peak load pricing and make people pay for those. When I have a business, which I do, and my capital runs out, I stop expanding. I can't just arbitrarily say I want to build more and more and more, and expand when the capital runs out. That is the same thing that should happen with people using electricity. If there is none left to use at peak load times then there should be no more use of it. And then Dr. Wyzga said--this is a quote: "In the face of uncertain supply, no industry will locate in a certain area." Well, I would like to speak from the heart here, because in my area with all these power plants going up around me, we are seeing a mixing zone. You can fly any day and see a mixing zone of varying depths in the atmosphere. We are seeing our air turn brown and orange and whatever other color. And why are we seeing this? The utility that services my particular area has a total generating capacity of about 1,000 megawatts. That's Southern Indiana Gas and Electric. In that same area that Southern Indiana Gas and Electric serves, we have Indianapolis Power and Light, which has a major electrical facility which serves Indianapolis. We have the Indiana-Michigan Electric Company which is building the plant in Rockport, which I mentioned earlier. We have the Public Service Company of Indiana, just 25 miles north of Evansville, which is going to have a 3,250 megawatt station when it's finished. It seems that, with the Clean Air Act Amendments of 1977 and the fact that economic sanctions are placed upon places that don't comply with the Clean Air Act standards, the economic arguments that he mentioned do not hold water because of one very significant thing. If our air is clean today and not clean tomorrow, and we have a non-attainment status as a result of that, and all the electrical production that's going on in the area which is dirtying our air, causing us to be on non-attainment status, is going to places like Fort Wayne, Indiana; Muncie, Indiana; Indianapolis, Indiana; and Plainfield, Indiana; we are not receiving any of the benefits from it. All we are receiving is the pollution. So it is very simple if you are going to look at things on a national level to say yes, we need to build more and more power plants to supply clean energy growth. But for those of us who are in the lower end of the valley, we don't want to see it happen like it has happened here in Pittsburgh. I took a drive up the Ohio River Boulevard yesterday and I've never been so appalled in my life! Thank you.

Dr. H. Spencer: I would like to announce some ground rules for this debate. First of all, I'm not involved in it. I am sure that many of you realize that that requires the exercise of some considerable restraint. Secondly, I feel that the presentation was a side of a controversial issue, or many such issues, and I think it would be appropriate if anyone who feels to be on the opposite side, be given equal time. The panel first, please.

Dr. R. Wyzga: I think the question probably was directed more at me than my colleagues, although if they want to take a stab at the answer I would be happy to let them. Let me first of all say that I did not argue that we should dismiss environmental protection. I think environmental protection should, in fact, be implemented when it is needed. All that I simply argued is that when we consider environmental control measures, we should look at what the total impacts are. There are environmental benefits; there are conservation benefits; but there are also some social impacts. I ask that we examine these social impacts, something that we have ignored heretofore. Secondly, I have not argued against conservation. I think that everyone agrees that conservation is necessary. What I am arguing for is some resolution of the dilemma we now face because we are working on one hand with, I guess, the incrementalism of environmental policy versus the need for some type of an overall comprehensive energy policy. We somehow have got to learn to resolve these two policy directions. I don't really know your specific area so I can't comment on it, but I will say that having excess capacity is something that no one wants, even utilities. It costs them money, and if there is one thing that motivates utilities , it is money, economics. And I would add that, as part of the measures to try and insure that we don't have excess capacity, we have Public Service Commissions and Public Utility Commissions. If they are doing their jobs correctly, and I think in many states they are doing their jobs correctly, it is they who examine the need for electricity or energy in a given area and try to ensure that these needs are in fact met. I think even they are frustrated and have difficulty in dealing with some of the environmental policies at the same time.

Dr. H. Spencer: Does anyone else on the panel wish to respond to the remarks of Mr. Blair? Who feels to be on the other side of that debate from the audience?

John Campbell, New York: I am from New York and I don't want to talk too much because my situation is a lot different from the Ohio River Basin. However, we do have in New York, purportedly, according to a lot of people, something like a 35% reserve margin, and the idea that that is surplus power, beyond what is needed to insure a reliable service, has stopped a lot of power plants.

In my state, and it is certainly one consideration that is important here, 45% of our electricty is generated with oil. It results in tremendously high rates in my area, having nothing to do with the capitalization of the utilities.

The capital portion of a utility rate base is not the entire rate base. It also has operations and maintenance and labor, and an assortment of other expenses. In particular in New York State, we spend about 1.2 billion dollars a year for oil. In the future, if new plants aren't constructed, whatever additional demand occurs in my state, however small, is going to be met with oil and it is going to be met at ever increasing cost. We've done a study on the economics of power in New York State and we found that if we build power plants in large numbers, the savings to our utility customers run into the billions of dollars.

What hasn't been seen is that the number of plants and the virtues of conservation are not entirely unmixed. We live in a world where we are affected by other people: by Arabs, by wars in Iran, and by a whole variety of other effects. We also burn a lot of oil in New York City, and it adds a tremendous amount to the sulfur burden in the city and on Long Island.

I haven't been exactly organized here but to sum up--when you are considering whether or not to build a power plant, you have to look at the entire picture--all the economic effects and not only one small facet of it, which is whether you have surplus power or not. The one-facet approach is a very obtuse way of doing energy planning and it has cost me and my neighbors in New York State well into the area of 2 billion dollars or so, and it is going to continue to cost them more in the next few years.

Dr. H. Spencer: The gentleman in the blue sweater is next, and then Dr. Blome, and then the two right in the middle. I'd like to ask the panel if there is any response to the previous comment that you would like to make. I would prefer that we try to conduct this as smoothly as possible, but it's probably not going to be easy. I'll give you about five minutes and that's it.

Dan Swartzman, University of Illinois School of Public Health: I've been debating with myself whether to get up here and say anything or not. I don't feel that I am particularly on the other side of whatever issue it is exactly that we are trying to put our finger on. In fact, I see myself on a parallel line with the gentleman from the Electric Power Research Institute, certainly heading in the same direction, hopefully lines that will never meet. The reason why I say we are heading in the same direction is that the question he addresses is an extremely important question for all of us to be asking ourselves. If there is any group of people who ought to be willing to examine the other side of the coin, it is those of us who consider ourselves environmental advocates. We are the ones who have been saying for many years that there is no such thing as a free lunch. It behooves those of us who spend some time doing this, particularly those who get paid to do it, as I do, to sit back and see what is the total impact of what we do and of what we are advocating. It is terribly important to look at all of the social costs, all of the both quantifiable and nonquantifiable societal impacts of the actions that we propose that society take. Because of that I have, in the past, advocated a number of times that environmentalists should be behind the push towards cost-benefit analyses. Once we are able to really get a handle on some sort of assessment of the benefits of the programs we advocate, we will be in a much better position to achieve the goals that we have been fighting for. My concern is the way in which this issue was addressed, not the fact that it was addressed. I think it has a very proper place in the program. I am concerned about a few points that were raised, and I would just like to briefly hit each of those points. I think, however, that I commend to those who read this record sometime in the future--I assume you are going to put it in a time capsule or something--I feel they will be capable of judging Mr. Wyzga's work on its own merit, and I am not going to presume to tell them how they ought to read that work. But I do have a couple of points that I think we ought to think about in the work. First is the concept that health is not a significant issue in the development of new power plants. The reason why a lot of the arguments have centered around visibility and esthetics and costs, is because we don't have a lot of the data we need to argue the health point. For instance, in the New Source Performance Standards that are being considered by EPA and debated by people in front of them, there is a concern for pollution from coal burning. I feel that is implicit, in where a lot of the environmentalists are coming from, is a concern for the health of our population. But since we don't have the sulfate information or the secondary impact, and we are stuck with a fairly poor indicator like sulfur dioxide, we are not in a position to be able to forcefully advocate the health side of these things. But I take fairly strenuous objection to the concept that the only thing we're debating is economics and esthetics, although I think those are extremely important, as one of the previous discussants mentioned. I also just want to point out that I didn't hear, during the presentation, any kind of association between hypothermia and higher energy costs. In fact, if I'm not mistaken, I heard, although it went by very quickly, that no association was possible at this time and so that all we have is that sort of anecdotal presentation that yes, in fact, it has happened. It is interesting to note, as was pointed out to me--this is not my thought-- that this supposedly has happened in England where we don't have the same sort of strenuous controls that we are asking the utilities to meet here in the United States. Also, I would like to point out to the representative of EPRI, and maybe he can take it back to his colleagues, that if, in fact, they're concerned about discriminatory burdens on individuals who have difficulty meeting high electrical rates, that maybe they ought to consider supporting rather than opposing inverted rate structures. That might get them to the same place that they're concerned about here. Also the gentleman pointed out that a number of the studies that he looked at were rather facile. After looking at his description of the situation in New York after the "blackout," I am quite certain as to his qualifications to make that judgment. I would just like to end by reinforcing what I said at the beginning. It is a terribly important question, and we lose all credibility as environmental health advocates if we don't examine this question, but I don't think this record reflects the kind of examination that is needed. Thank you for this opportunity.

Dr. H. Spencer: D. Blome is next, and I would like to have someone following that who would talk about some issues related to the other two papers, which were very good.

Don Blome, University of Kentucky: I would like to address this question to Dr. Morris. You showed some very impressive films concerning the safety of nuclear transport, at least on land surfaces. There is going to be an increasing amount of nuclear traffic, both spent fuel and new fuel, along waterways. Will you comment on the safety and potential hazards of nuclear transportation accidents with regard to waterways and the integrity of those canisters in water?

Dr. Morris: I guess the only comment I can make is that the Nuclear Regulatory Commission standards for the casks that the spent fuel is shipped in do specificy standards for immersion that they must meet. They have to be able to stand up to tests of being immersed in water.

Dr. H. Spencer: Next person, please.

Jerry Fields, Pennsylvania Power and Light Company, Allentown: I would like to respond to the gentleman from Evansville--a personal response; I don't want to respond for my company. In Pennsylvania a utility is required under the Public Utility Commission Act Section 401 to provide reliable, safe electrical generation. Also your reference that projections of utilities were way off base is correct. In the early 70's, our company, for example, suggested that the demand in the future would increase approximately 7% per year. However, this has decreased sharply and now we estimate about 2.5% demand increase per year.

You mentioned your local electric utility company is doubling its size. Our company is, also. Our investment over the first 50 years is about 2 billion dollars and at the present time we are building a 2-unit thousand megawatt nuclear reactor which costs also about 2 billion dollars. But one thing you must remember is that for each year a nuclear power plant, for example, is delayed, the construction and interest costs will be higher by about 150 to 250 million dollars. This will then be passed onto the customer. In addition to that, each day that this new plant is delayed, we will be using approximately 100,000 barrels of oil. It should be noted that with high energy prices for all fuels that we have right now there is an additional dependence on oil for gasoline, tires, clothing, and other substances. Since there is a shortage of oil we are compounding the problem by using oil for power. In addition, if a new plant is delayed we may have air pollution problems by continuing to use old plants which are allowed to operate instead of being retired. Even though there may be an additional amount, let's say 36% electric above the demand, you really can't talk about that per company. Most companies work on interconnection and they work by economic dispatch which means that if you can produce electricity for three cents per kilowatt per hour, and another company can produce it for two cents, you are not going to operate your plant. In the interconnection that our company is in, the Pennsylvania, New Jersey, and Maryland interconnection, there is a lot of oil. These are basically older plants. For example, Philadelphia Electric has a lot of oil units. That is one reason to go to new plants, possibly coal and nuclear. This means a utility, like our company, will be having excess electrical generation even though we prepare environmental reports for fossil and nuclear plants and simultaneously talk about conservation. For example, we talk about insulation, storm windows, purchasing appliances that use less electricity. So on one hand, we are building power plants, and on the other hand we are talking about the conservation. It seems like we are in a vice that we are trying to do as much as we can for our community, for the public, to make certain they have safe and reliable electricity. And yet once you start building a power plant, as it was mentioned, I believe this morning, it takes about 10 to 12 years for planning and construction. You just can't stop and wait a few years until electricity is needed. You have got to continue your programs and try to provide a good environment for the community living around the power plant, and also you have to try to give the consumer the best rate possible. I just wanted to give a little information on that. Thank you.

Dr. H. Spencer: Thank you. Now did anyone on the panel wish to speak to any of that? Dr. Radford, and Dr. Hamilton. Then I will try to assess the crowd at that point.

Ed Light, Appalachian Research and Defense Fund and also ORBES Advisory Committee: I feel there is a very important issue relating to the health effects of electric power transmission which wasn't covered, and I really feel that ORBES has an obligation to get information on this topic. I'm not sure that many members of the panel would be involved in this issue, but perhaps other people in the audience would have information and I would urge ORBES to go into this further with perhaps people who aren't here. This has to do with the effects of spraying herbicides under the electric power transmission lines. In West Virginia we have had complaints for several years from people all around the state concerning problems caused by this application. Up to now, it has been mainly 2,4,5-T used on these electric power transmission lines. The concern for health effects ranges from birth defects, skin irritation and cancer. EPA, in just the past weeks placed a ban on 2,4,5-T, which for years the power companies kept telling us was safe. Of course, the manufacturers of the chemical will challenge this and the decision could possibly be overturned. Probably more significant, in terms of what we ought to consider for the future, is that the companies will try alternative sprays, i.e. 2,4-D and tordon, which also would have a range of possible human health effects in addition to ecological effects. Another problem I have is with Dr. Phillips' presentation. He summarily wrote off the school of thought which has concluded that there are possible health effects, based on research up to date, from very high voltage transmission lines. I don't have any expertise in this area, myself, but again feel that ORBES has an obligation to get information from somebody with expertise from the other point of view on this. I'm not sure there's anyone here that can speak to that side of the question but obviously there is animal data and Russian research data to the other point as Dr. Phillips just said he disagreed with it. I would have liked to see him go into specifics as to why he disagreed with it. He didn't choose to do that. I'd like to make one observation on Dr. Wyzga's presentation. I was fascinated by the industry's willingness to accept the epidemiological work on the unemployment and blackouts question. Yet you talk about the epidemiologic work on sulfates and they're not quite so ready to accept it.

Dr. H. Spencer: I have already asked the panel members--perhaps I have not gotten to all of them. About the Agent Orange question, I'll give a one sentence response to that if they don't feel there's anything they can say about it. Dr. Phillips, would you like to respond to the question about the Russian data base in relation to what you said?

Dr. Phillips: First I will respond to the second question concerning other studies which have been conducted in the United States, and then answer the first question concerning the Russian data base. I did not have the opportunity in my 30 minute presentation to discuss the value and shortcomings of earlier studies that have been done in this research area. I did not mean to give the impression that earlier research has no value. This is not the case, of course. There have been several rather extensive critical reviews of past research. Some of these appeared within the last year in the open literature. It is not possible to discuss the rather massive amount of research that has been conducted on biological effects of ELF electromagnetic waves. It would require more time than we have available in this discussion period. In response to the first question concerning Soviet research in this area, during the past year I had the opporunity to visit the Soviet Union and talk with their scientists as a U.S. participant on two different U.S.-USSR exchange programs. I briefly summarized some of their findings in my presentation. I had an opportunity to talk to the group at an institute in Leningrad who published some of the work at the 1972 Cigre meeting, the group doing the animal experimental work at Kiev at the Communal Hygiene Laboratory and to several groups of scientists at institutes in Moscow. We can't ignore the Russian results. Data is data. I would be the last one to say that their data are not valid. Philosophically data is data; it's neither good nor bad. How you interpret the data is another issue. There are two potential problems with the Soviet research that has been conducted to date. First, they use experimental designs and procedures that are somewhat disparate from acceptable Western standards. For example, they do not always use appropriate control groups in their studies. This makes it difficult to determine whether the effects they observe (i.e. 60 Hz fields, or whatever) are the result of the agent in question or due to some secondary factor (i.e. temperature, humidity, noise, etc.). One starts to wonder how one can claim an effect when there is no appropriate control available to make such a claim. This situation was not evident at several laboratories in Moscow. Unfortunately, the results of studies from these laboratories have not been published yet. There appears to be some disagreement among various scientific groups within the Soviet Union concerning the validity of some of the earlier research on effects of 50-Hz electromagnetic fields. My present position is neutral. Let us wait till all the data are in before we make judgments. There will be a meeting with the Soviet scientists this June in Seattle, as part of this U.S.-USSR Exchange Program. Both U.S. and USSR scientists will present their latest results. We should have more information available at that time concerning the most recent Soviet findings.

Dr. H. Spencer: Did anyone else wish to speak to this issue?

Dr. Wyzga: I think there was a comment made about my not qualifying some of the results of those other studies. I think if you read my paper carefully, you will see that, in fact, I did qualify them. Professionally I am a statistician and I believe it is an obligation to my profession to scrutinize any result before I formally declare whether or not I believe it. I simply made observations of these results and indicated that they need scrutiny before we can determine whether or not we can accept them. Perhaps they generate hypotheses which should be tested in other studies.

Dr. H. Spencer: Dr. Radford and Dr. Hamilton; I do have a policy of trying to allow all persons to speak. If you already had something to say, I'm not going to come back to you immediately but I haven't forgotten that your hand has been raised.

Dr. Radford: I have a comment concerning the transportation issue, a question for Sam Morris, and then a further comment on what we can perhaps call a conservation/environmental argument. The comment relates to the earlier question about the shipping of wastes, coal or other materials by barges. I was a few minutes late coming in, Sam, and I may have missed it, but if you didn't comment on the use of river traffic for handling waste or fuels, I wish you would. It is my understanding that there are definite health costs involved in that, too. Barges do get loose and cause various problems. It is particularly relevant to this part of the country where a lot of fuel is transported by barge. The question I have has to do with your statement. If I interpreted you correctly, you said that the occupational accident mortality in the transportation of coal created more deaths than in the mining of coal. Is that a correct statement?

Dr. S. Morris: It depends on the situation. If you look at a strip mine situation, I think that is true.

Dr. Radford: You can qualify it if you like but I would just like to ask the question,: In reviewing that conclusion where do you see the preventive strategies? I mean, a lot of what we are discussing in this symposium has to do with historical things that have gone on now for the last several decades, and may involve technology that may be out of date. One of the things, obviously, we all hope to do by uncovering these problem areas is to prevent them. In other words, we don't have to accept the fact that, say, 5,000 men die each year moving coal around this country. In the future maybe there are things we can do and maybe that is part of the cost we are going to build into the fuel, too. That's what we are talking about when we protect the environment. So my question is, would you address that?

Now a final comment with regard to Ron Wyzga's presentation. What I am hearing is not so much the confrontation that some people seem to feel. In other words, I don't think that the separate courses are necessarily parallel, never to meet. It sounds to me as though what we are really saying here on this conservation versus environment argument is that, I think, we are almost at a point where we can uncouple environmental control and environmental costs from the ultimate societal cost or benefit of generating electricity. In other words, I don't see that there is a one-to-one correspondence between protecting the environment and the final cost that is going to go to the consumer. It is relevant to some of the things that the critics have just raised. I wish perhaps you might speak to that.

Dr. S. Morris: I appreciate your question very much because it gives me an opportunity to push a point that I try to make as frequently as I can. I think there are two criteria that we have to consider when we decide how much prevention we want to have. After all, just because we can calculate what the health effect is, really doesn't say anything about how much you should then do to try and reduce that health effect. Maybe it is already reduced to the maximum feasible amount. The first criterion is the total impact, and those are the numbers that I was showing, so we can compare how many deaths per gigawatt electric year in transport, as compared to mining, as compared to air pollution, and give us some idea of where the biggest effect is. That gives us some basis of deciding where we want to put our emphasis on prevention. But there is another criterion also, and that is the level of individual risk. I think that that difference is very clear when you compare coal mining with the transportation effects. Although our calculations show that the per gigawatt year total effect of transportation accidents and coal mining accidents are roughly the same, that is within a factor of two or three or something, the individual risk to the coal miner is much higher because the number of coal miners involved in producing that coal per gigawatt year is much less than the number of people who are at risk of being hit by freight trains. At least that is true on a national average basis. There are probably local situations where the individual risk in a local community that has a lot of unit train traffic is perhaps higher. I could envision that there are such local situations. One indication of that is that the Department of Transportation keeps records on railroad employees who are killed at grade crossing accidents off duty, in their private life, not occupational. It is hard to calculate a rate but there are enough of them so that the individual risk must be high. I assume it is just because those people live and travel around railroad crossings more than the average person. I hope that answers that part of the question. On barge transport, the data we have been able to look at from the coast guard on water transport would indicate that the deaths and injuries in accidents in barge transport are considerably lower than in railroad transport. There is quite a variation between inland river rates and Great Lakes transport, though. Great Lakes transport has a higher risk, particularly occupational risks. I'm not sure, offhand, of the difference in risk to the public, like small boats being run down.

Dr. H. Spencer: I would like just very quickly to answer Mr. Light on behalf of this question to Agent Orange. Mr. Light, the ORBES Project personnel will not let that go unnoticed. We are working on it. We have a good deal of data on the pharmacological effect of that compound which goes back to 1940 up to the present. Now Dr. Hamilton and then Dr. Zeller, and this gentleman right here, and I think John Blair, have been patient long enough. I'll recognize John if there is still some time left.

Dr. L. Hamilton, Brookhaven: I have several questions for Dr. Phillips. The first question is: Are the effects that you describe linked by some change in the membranes of cells and I wondered whether they are all recoverable or whether they are permanent? Have you any observations on the recoverability from these effects--in other words--their duration? That is the first question. The second question is this: In assessing the health and environmental impacts of energy we would like to include some quantitative data from this question of power transmission effects. So for this purpose it would be very helpful if you would tell us if you have done any thinking about the numbers of workers that might be exposed to these sort of effects, and whether or not the public is inevitably exposed, or whether they can be adequately protected by eliminating them from coming anywhere near these rights of way? I mean, what hazards are the public and occupational people really going to be exposed to? Thank you.

Dr. Phillips: Unfortunately, I could only spend 30 minutes to cover the tremendous amount of research. To date we have found very few effects. Of the many systems we have examined, the few effects appear to involve the nervous system, behavior, and immunology. Our plans in the immediate future are to investigate the relationship between effects and magnitude of field strength and/or duration of exposure, and to examine recovery from effects to determine whether such effects are transitory or permanent. Such data are not available at this time.

Concerning the mechanism of interaction of fields with biological tissue, I believe research on membranes may be a fruitful area. We currently have a study in progress that is addressing potential membrane changes by exposure to 60-Hz electric fields. These are in vitro studies--experiments in which cells are removed from the body, exposed to fields, and then studied. The major issues of concern in terms of human health are occupational and general public exposures.

With regard to occupational exposures, the population exposed to the largest electromagnetic fields are people who work in switchyards. Just recently, within the last year, some very valuable data have become available concerning the durations and intensities of exposure to some of these workers. Much of these data have not been available earlier because the worker is exposed to various field intensities at various durations of exposure throughout his workday in the switchyard. Recently, a personnel monitor has been developed by Dr. Deno at Project UHV which gives cumulative exposures within various bands of field strengths. Data they are now collecting with this new device should provide us with a better estimate of how long workers are exposed to electric fields and at what strength. Public exposure is normally very small. Only a few people walk under EHV transmission lines. These are the hunters and campers who sometimes follow the transmission line rights-of-way because it it easier to walk along these cleared areas. There are farmers, of course, who grow crops under EHV transmission lines. The general population, aside from those mentioned above, are seldom exposed to electromagnetic fields of EHV lines. There is not a good quantitative estimate of the durations and intensities of exposure to such groups as hunters and campers who walk the transmission corridors.

Dr. H. Spencer: Based on some recent literature, I think the membrane question is a dynamite issue. It truly is. Mr. Zeller, would you go ahead, and then John Blair.

Tom Zeller, Indiana State University: One point I just want to point out. We have heard a lot of epidemiological studies--animal studies--shot down for statistical reasons, etc.

and I just want to point out that works both ways. If the effects are derived from animal studies you can't necessarily conclude that there are no effects in humans. Currently the policy is that man-made substances have full constitutional rights and are deemed innocent until proven guilty. If the burden of proof is on the consumer, the only advantage I can see is that it makes for great material for epidemiological studies in the future.

My questions are as to the separation of conservation and environmental control-- that's a good point and I think we need to do that as much as possible, but I want to point out that there is one place in which they are really connected and you can't separate them. It's the whole issue of placing the true cost on the product, itself, instead of part of the cost of production being born as social costs and health costs, which is what this meeting is all about. Until you do that, I don't see how people can make true conservation decisions. When I flip on a light switch, I'm thinking I am creating air pollution and nuclear waste by doing this and I take that into account because I know that. But the general public doesn't know that, and the people in Fort Wayne, Indiana, do not know that when they turn on their switch, they are polluting my air due to the Indiana-Michigan Plant down near Evansville, Indiana. So that's one place where, until those environmental controls are put on that plant, those people in Fort Wayne are not being made to bear the full cost of consuming their product and therefore cannot make a reasonable conservation decision. But otherwise I agree with that point.

I would like to ask Dr. Morris about the impressive film of the transportation of radioactive waste. I am impressed by the efforts that have been made to contain these wastes during transportation, and I am not sure what else you can ask them to do. But I do want to ask, since we have been talking about statistical reliability of tests--how statistically significant were those tests? Did they do one casket a couple of times? Did they do it several times? How significant is one test under ideal conditions at the Sandia laboratory without taking into account human error? Was it statistically significantly done? Can you answer that?

Dr. Morris: I am not sure exactly how to evaluate the statistical significance of it. Those tests were in the context of a much larger testing program which involved computer simulation and scale model tests. I believe the ones shown were the only ones done of the full scale tests. You say that they are sort of ideal conditions. I don't really know that they were ideal conditions. I think they were purposely designed to be extreme conditions. In other words, most of the trucks that are carrying spent fuels are not going to be traveling at 80 miles an hour. It is very unlikely that they are going to find as solid a concrete wall to crash into as they did at Sandia. I don't think they are ideal conditions. I think they were done as sort of verification tests to determine whether the findings from the scale model testing and the computer studies actually held up in a full scale test. I'm sorry that, at least right now, I can't really give you a statistical evaluation of what that means.

Tom Zeller: I just wanted to ask Mr. Phillips about the effects of farming under those high voltage lines. Are there any problems with that or are there just no problems, so that you can go ahead and farm as normal?

Dr. Phillips: I can only answer that question from what I have read both in the popular press and from the very few scientific papers that have appeared in the literature. There have complaints by farmers, even in the area where I live. They complain of spark discharges. For example, they drive tractors under the transmission line, and may receive spark discharges when they get on or off the tractor under the line. There may be complaints in some areas by people who are doing farming, i.e., picking crops under a EHV transmission line, especially if they are using any kind of wire support for the crop. They may experience spark discharges. All the complaints that I am aware of have been directly related to the spark discharge problem, and of course, to the land utilization issue.

Dr. H. Spencer: The gentleman in the light tan coat? I'd like to ask the panel, to stay another five minutes. Is that all right with you people? There seems to be a considerable amount of interest here. Go right ahead.

Richard Ulrich, Group Against Smog and Pollution (GASP) Pittsburgh: The first comment is concerning Dr. Phillips' results that he presented from the Battelle labs. There were numerous effects that were not found in the studies so far but I think that it may be very important to notice that one effect can be enough if that effect is a serious enough effect. They have found two different things right now, the immunology and the nervous system. If these are serious enough, it doesn't matter that they have't found a list of 20 different things. I think this issue is reflected in other things that have been said, both this afternoon and earlier, about the size of the effects that are being measured or not measured. The animal studies, Dr. Phillips told me, involved usually about 20 animals. To see an effect with 20 animals, it sometimes requires a rather large effect. The effect of pollutants in epidemiological studies, partly because the pollutants, themselves, are measured very badly, and partly because the individual exposures are measured very badly, as Dr. Ferris said, from the Six-City Study where the indoor levels are quite different from the outdoor levels. My own assessment of the S02 data is that the health cost of S02 could be anywhere from nothing to 20 billion dollars. This is about the confidence limit that we can place on it, or maybe you could even put it on the other side, it could be helping 10 billion dollars worth. The size of these effects are not known and this relates to that kind of control. We don't know the cost of these health effects and the size they could be is a lot bigger than the point estimates that get made of them.

Dr. H. Spencer: Does anyone on the panel wish to respond to that? Dr. Philips, you're nodding your head aggressively.

Dr. Phillips: You addressed two very important issues, and we are acutely aware of these issues. Let me address the second question first, about the sensitivity of our tests. In the biological screening experiments, we are using a very broad, screening approach. If an effect is seen in a particular study, then we look at the system in much more depth. Each investigator from past experience in his research area, knows what size population he needs by having knowledge of the test sensitivity and variance. He will know how many anmimals he needs to show an effect, with 95% confidence. Of course, one has to be realistic and weigh cost and effort against the probability of detecting an effect with a certain test system. Some biological systems may be more sensitive than others. Other systems may not be very sensitive, partly reflecting the fact that some biological systems have high variability or are protected by compensatory mechanisms; this can "hide" effects. In our studies, each investigator is using the most sensitive tests he has to detect effects. This relates directly to your first question: whether one effect can be as important as, or more important than, 20? Of course. What a scientist must do is first to identify the effects -- what biological systems are sensitive. The existence of such effects have to be confirmed by others in independent studies. Once an effect has been found, you attempt to determine the consequences of the effect. This is, you start in-depth studies to obtain a better understanding. For example, in our study we see a change in synaptic excitability. This is an extremely sensitive test. This effect may not be reflected in the behavior or function of the whole organism. However, such sensitive tests as this may provide important insight into the mechanism of interactions of electromagnetic fields with biological tissue. Such information is needed to design specific tests for possible effects that may be harmful. Also, data from such studies may provide the foundation for epidemiological and clinical studies on humans. Eventually we need to examine either volunteers or occupationally exposed groups. This issue addresses a previous question from the audience: can effects occur in humans and not in experimental animals? Yes. The human species is very complex, especially behaviorally. What we do in laboratory studies on animals is model biological systems and, hopefully, obtain the basic information that is needed to relate this information to the human exposure situation. Eventually, however, you have to be able to relate research results from the laboratory to humans by conducting studies (assessments) on man.

Dr. H. Spencer: The gentleman at the fourth table here, and then Mr. Blair in overtime.

Jay Henry, Illinois Power Company: I'd like to just clear up something for the record as far as the gentleman from the Appalachian Research is concerned. It wasn't clear to me that it was pointed out exactly what happened with the two herbicides, 2,4,5-T and 2,4-D. It wasn't a proposed ban or anything like that. It was an absolute prohibition of using those two herbicides immediately effective when Barbara Blum made the announcement about three or four weeks ago. I'd like to point out that it was only the second time that the EPA has done something like this without any detailed statistical epidemiological studies. Clearly the EPA thought that there was enough evidence to take this type of action based on some observations that were made on miscarriages in women living in areas where these two agents were broadcast for "right of way" control. The ban went on a little further than just broadcast applications and completely banned the use of them. Our particular company has not been broadcasting herbicides for some 20 years now, although that is not the case with everybody. But it was an absolute ban and even removes the use of them from selected basal spraying. I think that's possibly unfortunate because these two agents might possibly be used in that type of application in an environmentally acceptable manner. But at any rate, it is a complete ban until further notice. I just wanted to clear that up. The other thing I would like to speak to, and this may surprise him a little bit, but many of the things Dan Schwartzman spoke about earlier, I am in complete agreement with. And I think it's good that the two sides, if you can categorize people into environmentalists and industrialists for the time being, their disparity in viewpoints is getting a lot closer together. The one thing I would point out to him is in a passing comment he made concerning utility rate structures; that we should get away from inverted rates. I'd like to point out that utilities aren't in favor of inverted rates just for the sake of being in favor of something. Rate structures supposedly have one overlying principle, and that is the cost of service to the customer. Where large customers can be served readily with a single transmission line, and they take up the distribution after it gets to their point, it costs a lot less to provide service. With respect to some of the other items in terms of "time of day metering," and load management, to try to spread out the peak loads and drop the peak load a little bit. Those types of things are being actively pursued by the industry, as well as institutes such as EPRI and others. I think that all these things are being considered. They are looking not only at what they do for the load, but also the cost of those types of services or those types of metering devices. The industry is doing its part in those areas.

Dr. H. Spencer: Allright, thank you. I'll add to that as John is coming to the microphone. EPA is concerned, as is the air force, particularly about the cockpit crew of the Ranchand Aircraft. These persons engulfed considerable amounts of Agent Orange during the Vietnam war and, of course, suffered.

Mr. J. Blair: A couple of points. One of them I would like to ask is about the transportation of nuclear waste. The subject was brought up about barge transportation. Well, the Hoosier National Forest has been mentioned as a possible permanent storage site for radioactive waste. And that sits on the Ohio River in Perry County, Indiana. What are the prospects for barge transportation of radioactive wastes? And if they use a cask--like the tow boat that recently sunk in the Ohio River near Evansville--that took more than two weeks to raise, what kind of contingency plans are involved in something like that?

Dr. S. Morris:: I am not sure that I can immediately answer the question. You want to know what kind of planning is done in case a barge were to sink?

Mr. Blair: Well, essentially the first question is: are barges one transportation mode for radioactive waste?

Dr. S. Morris:: Yes.

Mr. Blair: Okay. What kind of contingency plans? I can see these films and they are truly impressive and it does make me a believer. But it also makes me wonder about the human error that is involved in something like that, once one of these casks sink to the bottom of the Ohio River. Is it going to stay intact for the duration of the time it is going to take to raise it; does it take a couple of weeks or a couple of months?

Dr. H. Spencer: I might give you fair warning that Mr. Blair is a Pulitzer Prize winner in news photography and I think he is waiting. He is looking for a subject. You had best be careful with your answer.

Dr. S. Morris:: I can't say specifically what the contingency planning for something like that is. It would depend on the situation, I guess. As far as whether the cask would stay intact for the time it would take to raise it, I have every reason to believe that there is no difficulty there. But I can't say from any specific knowledge what difficulties there might be.

Mr. Blair: Okay. What prompted my question is the fact that originally we were told that it was safe to put this stuff in 55-gallon drums and dump it in the ocean. Then we were told it would be okay if we put it in storage tanks or, better, in underground salt formations. I'm just wondering, when the whole nuclear waste question is really going to be solved?

Dr. H. Spencer: Did you want to respond to that, Dr. Morris?

Dr. Morris: I think that at this point I don't necessarily want to respond directly to the question of when are we going to find a final resting ground for nuclear wastes. I might say, just on a specific point, that there has recently been a program at Brookhaven to try to recover some of the 55 gallon drums that were dumped in the ocean during the 50's. They have successfully recovered a few drums and the idea was to see whether there is any problem; whether the drums, in fact, leaked. The answer is no, they didn't. They didn't find any problem with the drums that they did recover. But that is just a sort of side-light issue.

Dr. H. Spencer: Dr. Hamilton, did you want to speak to this or some other issue?

Dr. L. Hamilton: Yes. I just wanted to speak to this issue because I think it is terribly important, first of all, to separate transportation from the question of ultimate waste disposal. I think those are two separate issues. I think that before anybody would decide on using a site, they would have to do a very thorough specific environmental analysis of all the routes to that site. I assume that would be part of the process. Now you mentioned several things that lead me to believe you are confusing a low-level waste with high-level waste. For example, the stuff that was dumped in the ocean in 55-gallon drums was low-level waste. They have never dumped high-level waste in 55-gallon drums in the ocean. And West Valley, or whatever it is, I always get confused about this place in New York, the problem there is of low-level waste. It is a low-level waste problem that they haven't been able to handle. I think the whole question of the storage of high-level nuclear waste in this country is something that hasn't been settled, as you point out. There never was a decision vis-a-vis Lyons, Kansas. There was a decision, if my memory serves me correctly, that the Atomic Energy Commission had decided to go to "over-ground engineered storage," just at the time Mr.

Schlessinger took over the agency. And then to show that he was a really big, tough administrator, he was going to shake things up. I believe this is a fair appraisal of that. He said, "No, we are going to start again." So he bears a very heavy burden of disrupting that particular analysis and decision. And the next thing that happened is, again if my memory serves me correctly, this question of underground storage at Kansas was raised as a possibility, but when they looked into the area, apart from the public opposition, the important thing is that it turned out to be not as geologically stable as they first thought. So it was never really seriously thought of as a solution. It was raised; there was a lot of public debate about it; but it was never put forward as a definite proposition. And there has been a further consideration, as you know, in the New Mexico area and those things are still under consideration. I think if you look at the latest report that has been issued by the Deutsch Committee, you will find that they say, very specifically, that before they choose any individual site, and they decide to go to that particular site, they are going to have to do some further corroborative measurements before they make the ultimate decision. I think that is the status of it--of the nuclear waste disposal problem. What we have had, unfortunately, is what happens frequently when you have a sort of changing bureaucracy and you have not exactly the best brains of the country concentrating on the problem of high level waste disposal from nuclear power plants because it has not been an overriding pressing issue. They have been able to cope with it on a temporary basis. I think everybody feels this has led, unfortunately, to a great deal of confusion in the public's mind. There has been a great misperception of what this whole thing is about. But I think that is really a record of bureaucratic bungling, rather than the technological problem. However, technological problems still do remain to be solved and demonstrated and shown. I think that is a very pressing issue.

Dr. H. Spencer: Allright, Dr. Radford, would you like to call it quits on this process?

Dr. E. Radford: I think we ought to put a lid on it and give our panel a chance to take a break. We are going to be discussing this very issue tomorrow. Bill Rowe will be here to talk about EPA's view, for example.

SESSION V: HEALTH PROBLEMS IN NUCLEAR POWER DEVELOPMENT

Wednesday morning, March 21, 1979

Moderator: Niel Wald, M.D.
Chairman Department of Radiation Health
Graduate School of Public Health
University of Pittsburgh

HEALTH EFFECTS OF IONIZING RADIATION

By
Edward P. Radford, M.D.

ENVIRONMENTAL EXPOSURES FROM NUCLEAR FACILITIES

By
E. David Harward

OCCUPATIONAL HEALTH EXPERIENCE IN NUCLEAR POWER

By
Robert B. Minogue

HEALTH EFFECTS OF IONIZING RADIATION

By

Edward P. Radford, M.D.
Professor of Environmental Epidemiology
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University of Pittsburgh
Pittsburgh, PA

HEALTH EFFECTS OF IONIZING RADIATION

Edward P. Radford, M.D.
Professor of Environmental Epidemiology
Graduate School of Public Health
University of Pittsburgh

In this presentation I shall restrict my remarks to the health effects of low doses of ionizing radiation, such as those associated with most medical uses as well as exposures occuring to workers in nuclear industries. In general these cumulative exposures are well below 100 rem, or about 50 times background or less.

The two effects of interest in this dose range are genetic mutations resulting from irradiation of the germ cells in the gonads, and cancer production from irradiation of cells distributed throughout the body. Genetic damage will be expressed only in the offspring of the irradiated persons; for some types of mutations these may be manifested several generations after the event. Cancer arising from irradiation, in contrast, affects directly the individuals exposed. Another difference between the genetic and somatic effects is that cancer (a somatic effect or one affecting all body cells except the germ cells) is usually life - threatening, whereas genetic effects may involve health impacts that range from mild disturbances in specific body functions to crippling though not necessarily life - threatening mental or physical disability.

These are some of the reasons that comparison of the health significance of cancer induction with the genetic effects of radiation is difficult. Another important factor is that we have virtually no direct evidence of genetic effects on man, most of the evidence being derived from animal experiments. In the case of cancer induction, on the other hand, we have a substantial body of evidence obtained from study of human populations exposed to radiation for various reasons.

I shall not discuss the genetic effects of radiation in detail, but it is important to note that whereas in the 1950's genetic effects formed the chief basis for radiation exposure limits to the general population, since the BEIR I report in 1972, genetic effects have been considered to be of less importance than cancer in terms of the total public health impact of radiation. This circumstance arises not so much because genetic risks are now perceived as less serious than was thought, but more because the cancer risks are recognized as more significant now than previously. The chief reason for the rise in risk estimates for cancer is the longer follow-up of exposed populations, which has brought to light those radiation-induced cancers with long latent periods to development.

Ionizing radiation is not only the best-documented environmental human carcinogen, but it also induces a wider range of cancer types than any other single known agent. We now have about 40 studies of radiation-induced human cancer which have shown an increased risk of cancers of many kinds. For each of the most important types of cancer induced by radiation we generally have several separate studies, which permits comparisons of the excess cancer risk per unit dose among these studies. By and large the different studies corroborate each other with respect to the particular cancer types under investigation, a circumstance that adds weight to the resulting risk estimates, especially in view of the fact that the populations that have been studied are of different ethnic backgrounds and the radiation exposure conditions have been different.

Current estimates indicate that about 1 to 3 percent of all cancers in the U.S. arise from background radiation exposure, on the basis that the linear no-threshold dose-response curve applies. One to six percent of new genetic mutations each generation are also ascribed to effects of background radiation. This quantitative similarity between somatic and genetic effects of background radiation is consistent with, but does not prove, the idea that mutation of somatic cells is an important step in cancer induction.

The sensitivity of various tissues of the body to cancer induction by radiation varies considerably, but no obvious generalizations emerge in regard to this sensitivity. Some cancers at sites known to be influenced by hormonal factors are readily induced by radiation, e.g., female breast and thyroid, but others are not, e.g., prostate and uterus. Tissues of mesodermal origin are generally resistant to cancer induction by radiation, but myeloid leukemia is an exception. Nor does the induction of cancer correlate well with the normal rate of cell proliferation; for example, the small intestine with its high epithelial turnover rate is relatively resistant to radiation-induced cancer. Cancers associated with suppression of the immune system are not especially radiation-sensitive, indicative that such suppression by radiation is not crucial to radiation carcinogenesis. Considerations such as these emphasize the complexity of the disease or diseases we know as cancer.

The major radiation-induced cancers are defined by their relative sensitivity to radiation induction, and by their natural frequency in the human population. Rare cancers that are highly sensitive to radiation induction may be less important that more common cancers that are only moderately sensitive to radiation. Thus far, five types of cancer are the major factors in the cancer risk from radiation exposure. These are cancers of the female breast, thyroid and lung, leukemia and cancers of the alimentary tract, especially of the stomach and colon. In addition, there are a number of cancers whose induction by radiation is well-documented, but the risk is relatively less important. These are cancers of the pharynx, salivary glands, liver and biliary tract, pancreas, urinary tract, nervous system, bone, skin and the lymphomas. For an additional group of sites the magnitude of the radiation-induced cancer risk is uncertain. These include the larynx, ovary, uterine cervix and connective tissues. Finally there are a number of cancer types for which there is no evidence of an effect of radiation, including cancers of the prostate, testis, corpus of the uterus, the mesentery and mesothelium, and chronic lymphatic leukemia. The fact that such a large array of types of human cancer have been shown to be induced by radiation suggests that at high enough exposure and with long enough follow-up essentially any cancer can be shown to be induced by radiation.

Fetuses irradiated in utero are highly sensitive to development of certain cancers, but this increased risk persists after birth only until about the age of puberty. After a brief radiation exposure to children or adults, granulocytic and acute lymphatic leukemia as well as osteosarcomas (bone cancers) induced by radiation have a short minimum latent period to appearance of the cancer of two to five years, with eventual dying out of the effect in about 25 years. In contrast all other radiation-induced cancers in adults have minimum latent periods of about ten years or more, and with follow-up to the present, excess cancers continue to appear as long as 50 years after radiation exposure.

The excess cancer risk from radiation is not necessarily proportional to the "natural" risk, that is the relative risk model does not apply in general, but the effect of age at irradiation or age at cancer development is very important, an observation suggesting that radiation enhances or triggers the action of age-specific factors related to cancer induction, factors which are widespread in the population. Figure 1 shows a schematic way of explaining the concepts of absolute and relative cancer risks, as well as the concept of "expression time," that is the time that the excess risk is present during the life of the irradiated person. In each graph the curved solid line is taken as the "normal" or spontaneous cancer rate related to age. The upper graphs illustrate the change in risk for a cancer, such as leukemia, which is expressed only for a limited time. After irradiation and a minimum latent period, the cancer rate in the irradiated population then deviates from the spontaneous rate, but eventually returns back to it. This difference in cancer rates permits us to calculate the increased lifetime risk for that particular radiation dose. The right upper graph shows that if the individuals are irradiated at an older age the risk may be greater, that is, proportional to the spontaneous risk at that age. In this sense the risk is "relative," in that it is proportional to the spontaneous rate.

The two lower graphs illustrate the case where the expression time is assumed to last for the lifetime of the irradiated population. As in the first graph, upper left, irradiation is at a young age. In one case, at the left, the risk is assumed to stay constant, in terms of an elevated cancer rate, throughout the rest of life. In the other, on the right, the risk is elevated in proportion to the natural cancer risk. This latter model of the cancer effect is called the "relative risk model" and gives higher estimates of the cancer risk than the former, called the "absolute risk model."

Age at irradiation is thus an important factor affecting radiation risk. Figure 2 shows this effect for the Japanese A-bomb survivors. On the right, the excess cancers per unit radiation dose is plotted against age at irradiation. The excess for leukemia is shown as well as the excess of all cancers except leukemia. For leukemia the risk is high in childhood, decreases to a minimum and then increases with advancing age. A similar pattern is shown for other cancers. On the left the risk relative to the spontaneous rate is shown. This decreases from the childhood period and then remains relatively constant, indicating that the relative risk model is appropriate.

Figure 3 shows the same type of information for a group of British patients with an arthritic disease, ankylosing spondylitis, given deep x-ray therapy to the spine for treatment of their symptoms. These patients have now been studied for over 20 years, and have been found to have an increase in cancers of various types. This increase is shown plotted against age at irradiation, and also relative to the spontaneous rate (observed over expected). A pattern similar to the Japanese A-bomb survivors is evident.

Recent evidence from human follow-up studies continue to be consistent with the dose-response relationship for cancer induction being linear without a threshold; new data showing excess risk of several types of cancer are now available in the range of doses from a few rads to 50 rad. The evidence for a linear dose-response curve for leukemia is less certain. Women have a greater total radiation-induced cancer risk than men because of their greater sensitivity to breast and thyroid cancer. We have reason to suspect, moreover, that some subsets of the population are more sensitive to cancer induction by radiation, but the significance of such groups on dose-response data for whole populations is not known at present.

Figure 4 shows recent dose-response data on lung cancer in Czechoslovakian uranium miners, with excess cancer per 1000 miners plotted against radiation exposure in terms of "working level months." This unit expresses the product of the duration of underground exposure in months, times the concentration of daughters of radon gas present at the work sites, given in a standard unit called the "Working Level." It is the short-lived daughters that irradiate the bronchial tissue with alpha radiation and initiate bronchial cancers.

If you look carefully at the points at the lower doses, you will see that they can be fitted with a curve that bends over slightly and is concave downward. That is, lower doses are somewhat more effective per unit dose than the higher doses. It is also of interest that recent data from other miner groups and the Japanese A-bomb survivors indicate cigarette smoking acts primarily to shorten the latent period to onset of bronchial cancer, and that the combination of smoking and radiation exposure leads to a cancer risk that is not much more than additive for those two cancer inducers.

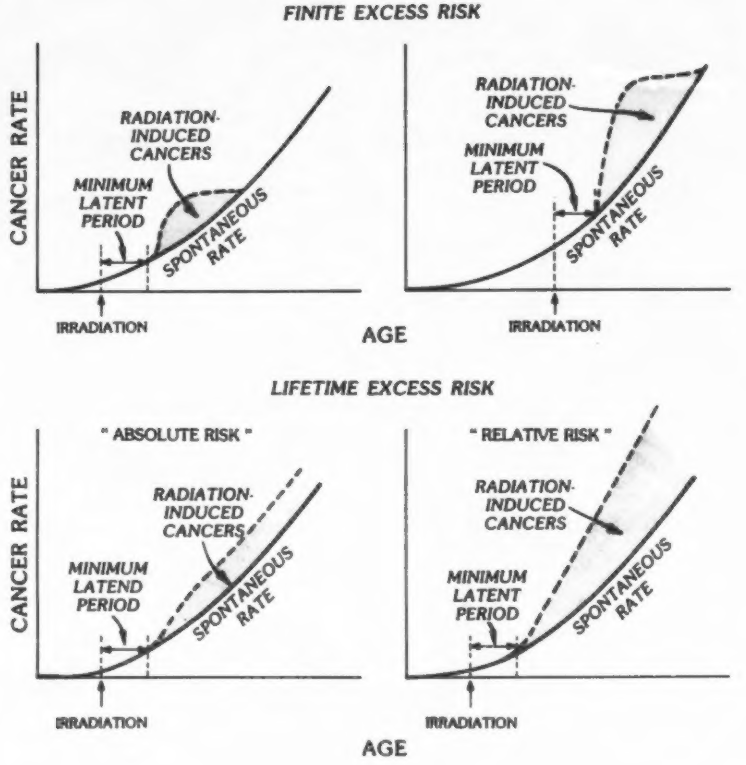
Finally, I present dose-response data for the Japanese A-bomb survivors, primarily because the information at low doses of radiation is quite extensive. Figure 5 shows the data for leukemia in Hiroshima obtained from the Leukemia Registry. Excess risk is plotted against the mean dose to the bone marrow, with a correction to take account of the greater cancer-producing effect of neutrons compared to gamma rays. The best fit to the data in the lower dose range is a line with a slight upward curvature.

Figure 6 is the same plot for the Nagasaki survivors, with identical lines drawn through the data points. Because the study group in Nagasaki is so much smaller than in Hiroshima, the error limits for Nagasaki are larger, and thus the precise dose-response relationship is less certain, but the curve in Figure 6 is identical with that for Figure 5.

Figure 7 shows the dose-response data for the incidence all the major radiation-induced cancers in Hiroshima and Nagasaki for the period 1959-1970, or from 14 to 25 years after exposure. The straight lines are weighted regression lines, and fit the data well.

In summary, we now have have a substantial body of human data which permits us to estimate cancer risks from irradiation, at least under conditions similar to those of the groups that have been studied. Our knowledge of possible genetic effects of radiation is limited to extrapolation of results from animal studies, but the numerical estimates of risk at low doses are similar for those of cancer. In both cases we apply the linear, no-threshold dose-response curve, not because the data conclusively prove its applicability, but because of experimental support for this model and because no other approach fits the facts better at this time.

FIGURE 1



FINITE EXCESS RISK

LIFETIME EXCESS RISK

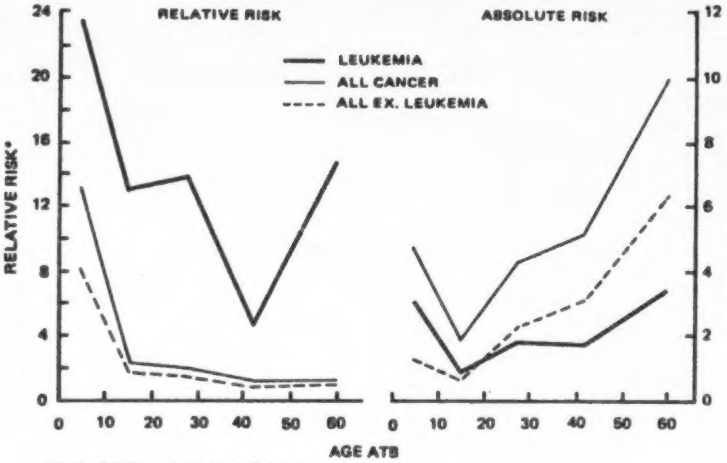
Some models of the effects of radiation exposure in relation to age. In each of the four graphs, the spontaneous cancer rate for a group is assumed to increase progressively with age, with the total age range covering the lifetime of the individuals exposed.

Upper graphs, Left: Irradiation at a young age, and with the excess cancer risk increasing and then decreasing after a period of time (finite excess risk model). After a minimum latent period, usually several years, before any cancer excess is observed, the excess number of cases is given by the area under the dashed line.

Right: Irradiation at an older age. The same time course of excess cancers is observed but a greater effect is present because the older individuals are more susceptible to radiation-induced cancer.

Lower graphs: Two possible results for radiation-induced cancers which have an increased risk lasting for the individuals' lifetime. Left: After the minimum latent period the cancer rate rises above but parallels the spontaneous rate. Right: After the latent period the cancer rate increases in proportion to the rising spontaneous rate. The former situation implies that the excess risk does not change with the rising sensitivity to spontaneous development of cancer, and is sometimes referred to as the "absolute risk" model. The latter case implies that those factors responsible for the increasing spontaneous cancer rate are also active in enhancing the radiation effect, and is referred to as the "relative risk" model. We do not yet know which of these two models may apply for those cancer types that may show a lifetime increased cancer risk after irradiation.

FIGURE 2



*Risk of 100+ rad relative to 0-9 rad

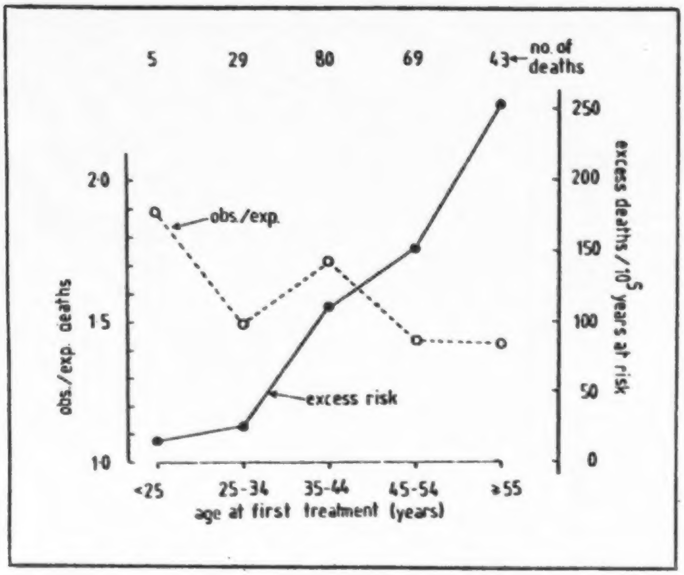
Effect of age at radiation exposure on the subsequent cancer risk. Data for leukemia (heavy solid lines), all cancer (light solid lines) and all cancers except leukemia (dashed lines) for survivors of the atomic bombing of Hiroshima and Nagasaki. Followup 1950-74. The data are plotted against age at the time of the bombing (ATB) in 1945.

Right graph: Excess cancer deaths per million person-years per rad for all exposed groups. Both for leukemia and for all cancers except leukemia the minimum excess risk is observed in the 10-19 age group, and rises progressively with age, in accord with the spontaneous rate among Japanese.

Left graph: Cancer risk for the heavily exposed group (100+ rad) relative to the risk for the low-dose group (0-9 rad), equivalent to a control group. For all cancers, the relative risk is highest in children and remains comparatively unchanged in adults, although for leukemia there is a suggestion of a minimum among the 35-50 year-old group. These results support the relative risk model at least for the effect of age at the time of radiation exposure.

Data from: Beebe, G.W., Kato, H. and Land C.E. "Mortality Experience of Atomic Bomb Survivors, 1950-74." Radiation Effects Research Foundation, Life Span Study Report 8, 1977.

FIGURE 3



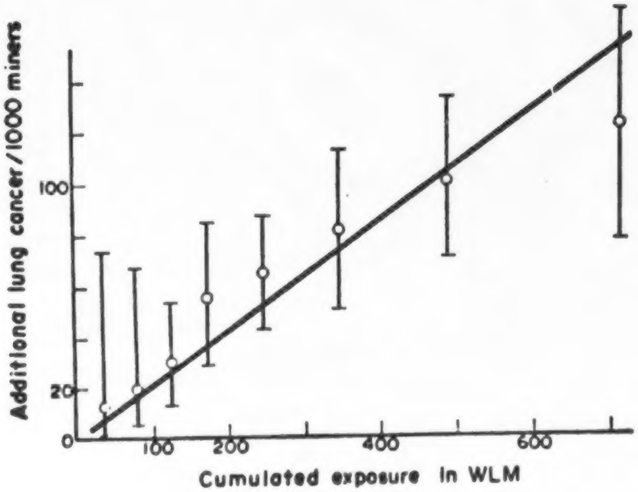
Effect of age at irradiation on subsequent cancer risk. Data are for British patients with ankylosing spondylitis given x-ray treatment to the spine and pelvis 1935-54. Follow-up to 1970. Data are plotted by age at time of x-ray treatment; number of observed deaths along top of graph.

Closed circles and solid line, right ordinate: Excess cancer deaths per 100,000 person-years at risk. The excess risk rises progressively with age at exposure, in the same way that the spontaneous rate in the general population does.

Open circles and dashed line, left ordinate: Ratio of observed number of cancer deaths to number expected from age-specific rates for British population. The relative risk is highest for the youngest group, but is relatively constant for the older groups. Again the data support the relative risk model applied to the age at irradiation.

Data of Smith, P.G. and Doll, R. Age and time-dependent changes in the rates of radiation induced cancers in patients following a single course of x-ray treatment. Proc. Int. Atomic Energy Agency Symposium on late biological effects of ionizing radiation. Paper 1AEA-SM-224/711 Vienna, 1978.

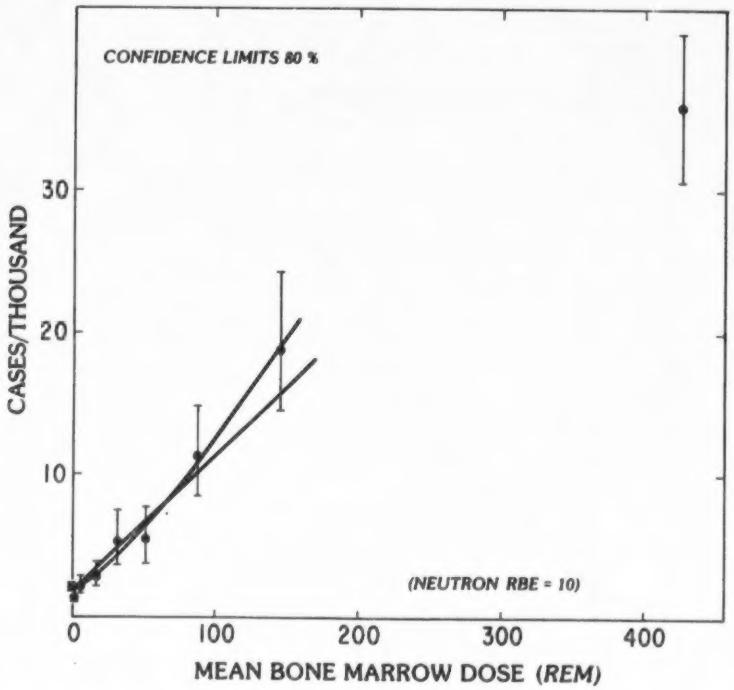
FIGURE 4



Excess lung cancer risk from inhalation of radon daughters. Czechoslovakian uranium miners studied from 1950 to 1973. Ordinate: Excess lung cancers/1000 miners. Abscissa: Exposure to inhaled alpha radiation in units of "Working Level Months," a unit which takes account of the radon daughter concentration times the number of months of exposure during the work. Error bars represent 90% confidence limits.

Data of Ševc, J., Kunz, E. and Plaček, V. Lung cancer in uranium miners, and long-term exposure to radon daughter products. Health Physics 30: 433-437, 1976.

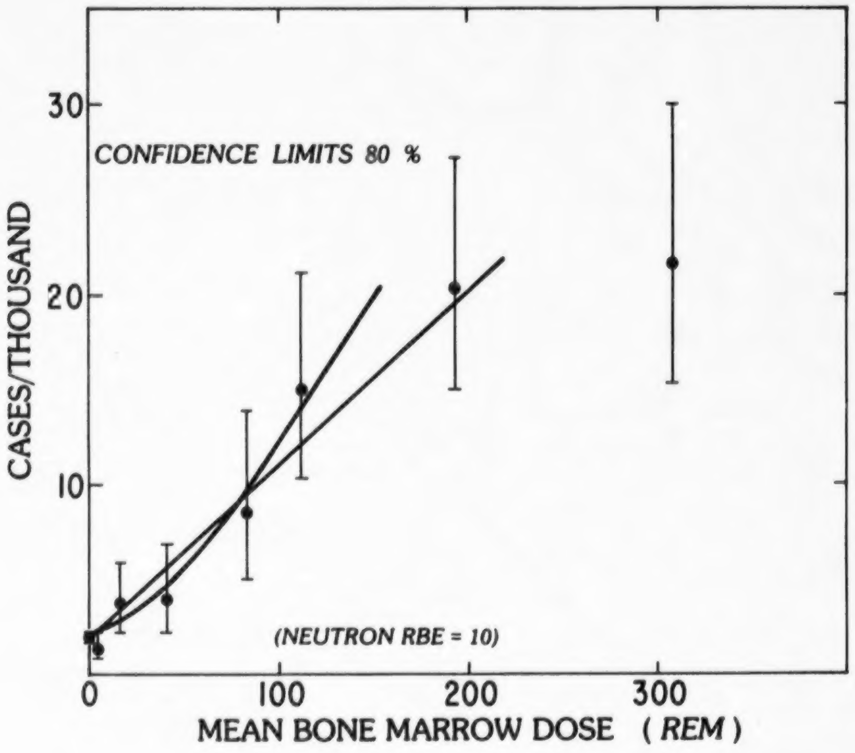
FIGURE 5



Dose-response relationship between radiation exposure and leukemia risk, Hiroshima atomic bomb survivors, Leukemia Registry data. Abscissa: Mean bone marrow dose in rem, with assumption of a quality factor (RBE) of 10 for the neutron component of the exposure. Error bars are 80% confidence limits for each point. Ordinate: Leukemia rate, cases per thousand exposed. The straight line is fitted by eye, as is the curved line, which appears to fit the data better. The point for the highest dose is not included because it is probably in the dose range where cell-killing may reduce the leukemia risk per unit dose.

Data from: Beebe, G.W., Kato, H. and Land C.E. "Mortality Experience of Atomic Bomb Survivors, 1950-74." Radiation Effects Research Foundation, Life Span Study Report 8, 1977.

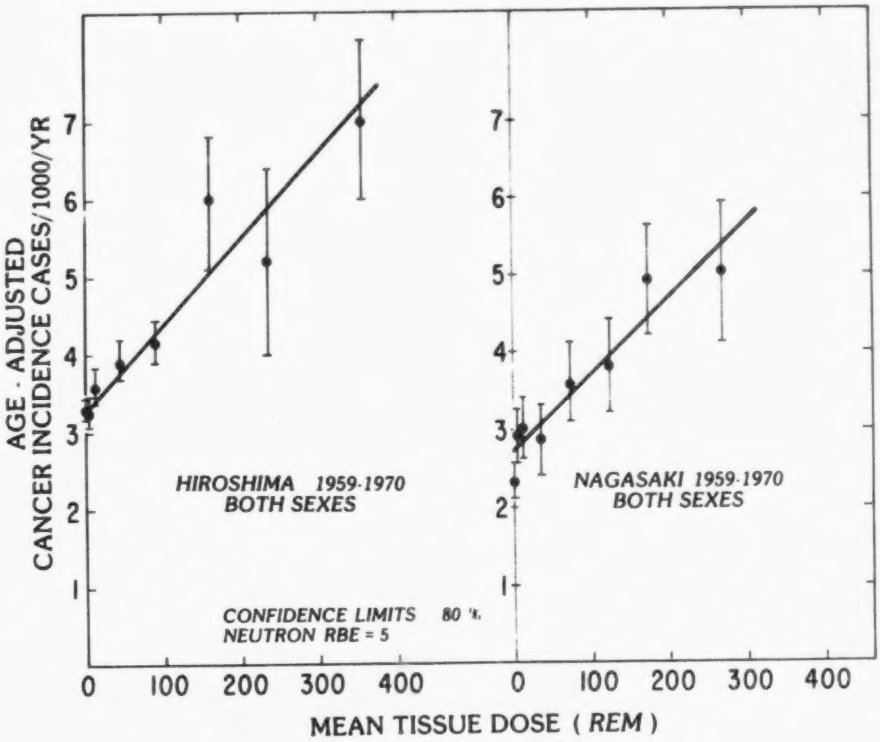
FIGURE 6



Same data for leukemia risk in Nagaski as for Hiroshima shown in Figure 5. The error limits are larger because the Nagasaki study group is much smaller than that for Hiroshima, but the same curve as for Hiroshima fits the data reasonably well. The neutron component of the Nagasaki bomb was small compared to the Hiroshima bomb, but application of a quality factor of 10 for neutrons appears to be suitable to bring the data for the two cities into consonance within the reliability of the data.

Data from: Beebe, G.W., Kato, H. and Land C.E. "Mortality Experience of Atomic Bomb Survivors, 1950-74. "Radiation Effects Research Foundation, Life Span Study Report 8, 1977.

FIGURE 7



Dose-response relationship between radiation exposure and total cancer incidence for both Hiroshima and Nagasaki. Data from tumor registries in both cities, but ascertainment of cases more complete for Nagasaki than for Hiroshima. Followup 1959-1970. Abscissa: Mean tissue dose in rem, with assumption of a quality factor (RBE) for neutrons of 5. Ordinate: age-adjusted cancer incidence, cases/1000/year. Error bars are 80% confidence limits for each point. Straight lines through the points are weighted regression lines.

Data courtesy of Dr. C.E. Land

ENVIRONMENTAL EXPOSURES FROM NUCLEAR FACILITIES

By

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Environmental Projects Manager
Atomic Industrial Forum

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Environmental Exposures from Nuclear Facilities

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Environmental Projects Manager
Atomic Industrial Forum

Presented at the
Symposium on Energy and Human Health:
Human Costs of Electric Power Generation
Pittsburgh, Pennsylvania
March 19-21, 1979

After Dr. Radford asked me to speak at this conference the other day, I started looking for the data which I thought might best represent the environmental radiological impact of the nuclear power industry. I came to the realization that radiation protection now suffers from paper proliferation the way everything else does with the enormous quantities of data now available. At that point I also wondered whether I had contributed to the current flood of data in my former role of bureaucrat with the Public Health Service and EPA. In a paper before the 1970 Annual Meeting of the Health Physics Society, I had uttered the phrase"...It is my opinion that some of the public relation problems the nuclear power industry is now facing could have been lessened if early in the game detailed data from operating nuclear power plants had been made available to the health and scientific community for interpretation to the public in terms of radiation dose to people."

Now hopelessly caught up in a web perhaps partially of my own spinning, I will attempt to bring you a perspective as to the radiological impact of the generation of electric power by nuclear energy. None of the data I am using have originated with the nuclear industry; it is largely from the Environmental Protection Agency, the Nuclear Regulatory Commission and the recent draft Interagency

Radiation Task Force Report by the Department of Health, Education and Welfare. At the risk of involving you in a numbers game, I feel compelled to use these data as the only way in which such an impact can reasonably be demonstrated. Qualitative comparisons are helpful but generally inconclusive. Both quantitative and qualitative analyses are, of course, open to argument. I have attempted to use data that are representative and I believe, within the range of values that are accepted by the majority of the knowledgeable scientific community.

I would like to show you a table that hopefully will help place in perspective the total contribution of nuclear power generation and its component parts to the total United States population radiation exposure. The sources and the corresponding cumulative doses in person-rems per year are from a table in the recent HEW draft report of the Interagency Task Force on the Health Effects of Ionizing Radiation.[1] I have added two columns: potential health effects and percent of total U.S. cancer deaths that might be attributed to various sources of radiation using the linear non-threshold concept. A conversion factor of 100 potential health effects for each million person-rems was used.[2] Please note that I carefully use the word "potential" when speaking of health effects throughout these remarks. From this table, perhaps one percent of the total of 390,000 U.S. cancer deaths per year might be statistically attributable to radiation, of which roughly half might be natural background related and nearly half related to the healing arts, largely from the use of diagnostic X-rays. I say this realizing there may be certain synergistic effects that are not yet fully understood between radiation and other substances.

My remarks will be addressed to the nuclear power industry component of this table which might account for some one-thousandth of the total radiation-induced health effects to the general public.

I would like to review the environmental radiation protection standards and regulations that are applicable to the nuclear power industry. Perhaps after this discussion, when you read the many reports of federal agency overlaps in the control of radiation, you will have a better understanding of what the problem is.

Table 2 shows the present basic U.S. radiation protection guidance for the public developed by the former Federal Radiation Council whose responsibilities were given to EPA with formation of that Agency in 1970.[3] These guides, in existence for many years, are used by all federal agencies involved in protecting the general public from radiation. Guides are also available for occupational radiation exposure.

Table 3 shows the NRC objectives for light water reactor waste treatment system design, Appendix I to 10 CFR Part 50.[4] These guides were promulgated in 1975 after some three years of public hearings. The NRC implements these numerical values as operating limits in the technical specifications of the license for each nuclear power plant in addition to reviewing the capability of radwaste treatment systems design during the licensing process. In addition, NRC regulates licensees according to 10 CFR Part 20, Standards for Protection Against Radiation.[4] Part 20 contains the detailed regulations which must be followed by a licensee during operations to assure public health and safety. Accompanied by detailed isotopic analyses of effluents, a comprehensive environmental surveillance program and reporting mechanism is provided.

After some seven years of development, the Environment Protection Agency in 1977 promulgated their environmental standards for the uranium fuel cycle (40 CFR 190) which are being implemented by the NRC.[5] These are shown in Table 4. These standards are effective December 1, 1979, except for uranium milling operations (December 1, 1980) and krypton-85 and iodine-129 which are effective January 1, 1983. These latter two standards (krypton-85 and iodine-129) apply generally to the fuel reprocessing component of the nuclear fuel cycle which, of course, is currently non-operable under U.S. policy.

Another applicable EPA standard is the regulation for public drinking water which is shown in Table 5.[6] This is a regulation to be implemented by state authorities but has been incorporated into NRC model radiological technical specifications as guidance to NRC licensees.

Table 6 shows two EPA authorities that are potentially of real significance. Another layer of regulation was applied to many industries including nuclear power in the Clean Air Act Amendments of 1977 when radiological provisions were included for the first time. The inclusion in the amendments of radiation was not well-known at the time but the wording of the Committee report made it clear that it was intentional.[7] EPA is now in the process of determining how to implement this Act and certain administrative actions must be taken by them by August, 1979. Hopefully, NRC will be the regulating agency for this Act under EPA auspices in the case of nuclear facilities; this appears to be the direction things are headed. However, the Act also makes it possible for states to control air emissions from these facilities, undoubtedly a complicating factor. One state has already taken initial steps to control such emissions.

Finally, the Federal Water Pollution Control Act contains provisions for providing water quality guidance to states.[8] An example of the regulatory complication that could result is a recent radioactivity guide for Great Lakes water developed by EPA and the Canadian authorities under the aegis of the International Joint Commission.[9] A radioactivity objective of one mrem per year was written for Great Lakes water based on a consumption of 2 liters per day. This objective potentially can be used by states under FWPCA to establish water quality criteria, thus inserting an additional regulatory layer.

Now that we have reviewed the various regulatory controls imposed on nuclear facilities, let's look at some of the estimated radiological impacts associated with the various components of the nuclear fuel cycle listed in Table 7.[10] These are listed in their general order of occurrence during the operation of the fuel cycle. You can see that over half of the impact might result from fuel reprocessing which, as I mentioned earlier, is not operating at the present time. The next highest impact results from the mining of uranium, primarily through the release of radon. Radon also is the major emission from the tailings resulting from milling operations. If one uses the linear concept, the potential health effects for each 800 MWe reactor and its part of the supporting total fuel cycle are about 0.056 per year although this number probably needs additional scrutiny...It might be slightly higher. If one extrapolates this to the current number of operating nuclear plants in the U.S., there is very close agreement with the HEW draft report table which indicated some 5.6 potential health effects in 1978.

It is of interest to discuss several specific radionuclides resulting from the production of nuclear power because of their unique properties and characteristics. The first radionuclide in this category is radon-222, a naturally occurring radioactive gas having a half-life of 3.8 days. It has been estimated that some 100 million to 240 million curies are released to the U.S. atmosphere each year, normally through the earth's crust. Many factors influence the exposure of people to radon. Homes built of concrete, stone or brick can have concentrations of radon three to seven times greater than typical outside air. Things such as agricultural practices, closing of windows in homes, home insulation, whether the home uses natural gas or not (which contains radon) may influence the levels of radon to which one is exposed Dr. Leonard Hamilton of Brookhaven National Laboratory[11] has estimated incremental doses from the fuel cycle using doses to the bronchial epithelium reported by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)[12]. He has estimated that the one year's operation of a 1,000 MWe nuclear power plant at a capacity factor of 65 percent would result in an increased dose to the bronchial epithelium of less than one-thousandth of a mrem/year (2.5×10^{-4}) from the mining and milling of uranium to fuel the plant. If one follows through Dr. Hamilton's assessment, the risk of developing lung cancer from naturally emitted radon is about 1.5×10^{7} times greater than developing lung cancer from radon resulting from the fuel cycle.

These data were presented before an NRC Atomic Safety and Licensing Board at a hearing on the Perkins Nuclear Station in May, 1978. The Board ruled that the release and impact of radon-222 associated with the uranium fuel cycle were insignificant in striking the cost-benefit balance for the Perkins Station. Nevertheless, there does remain some concern about the national average impacts of radon resulting from man's disturbances of the earth's surface from various activities such as mining of minerals EPA did not include radon in their uranium fuel cycle standard mentioned earlier. However, that Agency is evaluating radon from the standpoint of the Clean Air Act Amendments and the story on that still remains to unfold. It should be mentioned that the radiological impacts within close proximity of uranium mills are of more concern because of the presence of tailings, and considerable effort is underway by NRC to control this source of radiation exposure through stabilization of the tailings. I did not have available to me a new report which should be issued shortly by NRC which was prepared by Oak Ridge National Laboratory.[13] This report will assess the radiological impact of radon-222 from uranium mills and other sources and should be an important reference on this subject and may clarify some of the uncertainties in this area.

Another radionuclide of some interest is carbon-14 which is produced in nuclear reactors, largely in the fuel, but also in primary coolant. This nuclide may be potentially of greater dose significance than krypton-85 for which EPA has established a standard. However, since it is largely released during fuel reprocessing which is not now being performed, there would be time to more fully evaluate control techniques.[14] Although there is no present cost-effective control technology for carbon-14 removal, it is belived that certain existing krypton-85 recovery systems can be used for its removal. There has been some concern about the cost-effectiveness of krypton-85 control technology.[15]

However, this doubt may completely vanish if these systems are shown to have the capability for removing carbon-14 which is of potentially greater dose significance. Carbon-14 and radon-222 achieve their importance despite low per-capita doses largely through the exercise of multiplication of these extremely small doses by billions of people over long periods of time. Some people believe it is necessary to consider these materials for long periods because today's activities in the fuel cycle result in continuing releases of radon-222 and the persistance of the long-lived carbon-14.

I would like to leave with you some comments on the high-level radioactive waste issue. Although Dr. Rowe will be addressing this subject in depth later in the program, I think those aspects relating to the radiological impact should be mentioned in this review of nuclear facility impacts. EPA is now in the process of completing a detailed impact analysis of high-level waste disposal in connection with the development of an environmental radiation standard. To my knowledge, it is the most detailed analysis undertaken to date. They have evaluated the total radiation impact over a period of 10,000 years in a geological formation under very pessimistic assumptions and have found that there might be a potential 100-1,000 health effects as a "high value" over that period of time, with a "low value" four orders of magnitude less. These were the numbers reported to the House Subcommittee on Energy and the Environment by EPA staff in January.[16] Discussions with EPA staff personnel since that time have indicated that their "high value" is probably closer to 50-100 potential health effects over 10,000 years or somewhere in the order of one one-hundredth of a health effect per year.

Furthermore, EPA has reported what everyone with a knowledge of plutonium behavior has known, that plutonium is highly immobile in geologic formations and, as such, contributes less than one percent to the total health impact of high-level waste disposal. Thus, one of the most rigorous analyses available has shown that geological storage of high-level wastes can be achieved safely even under adverse conditions, although all efforts will be made to choose the safest site possible. It appears that the major remaining hurdle is to somehow get the U.S. waste disposal project moving in a forward direction.

In closing, I would like to state that much has been done to make nuclear power safe from an environmental and health standpoint. The potential impacts are small and indeed at least as small as other electrical energy alternatives. In this connection, it is my opinion that comparative risk analyses of the various energy options should be continued and refined. From my experience in environmental health, I believe that nuclear will come out well. However, whatever options are chosen to generate electricity, I am convinced that this nation will need the energy and need it badly in the near-term as well as the long-term and is in every sense of the word a public health benefit. The continued protection of the public health in the process is uppermost as a priority, but the general welfare of the public is also important, and that is coupled with the availability of energy at a reasonable cost. I believe the health impacts of all viable forms of energy can be made acceptable, but without the necessary energy available, many people both here and abroad may suffer needlessly.

TABLE 1

U.S. GENERAL POPULATION EXPOSURE ESTIMATES - 1978

Source	Person-Rems Per Year (Whole Body)	Potential Health Effects	Percentage Of Total Cancer Deaths
Natural Background	20,000,000	2,000	0.5
Technologically Enhanced	1,000,000	100	0.025
Healing Arts	18,000,000	1,800	0.46
Nuclear Weapons			
Fallout	1,000,000-1,600,000	100-160	0.025-0.04
Weapons Development, Testing and Production	165,000	16.5	0.004
Nuclear Energy	56,000	5.6	0.0014
Consumer Products	6,000	0.6	0.00015
Potential Health Effects Total		4,023-4,083	
Number of Annual Cancer Deaths		390,000	

Adapted from draft report of the Work Group on Radiation Exposure Reduction, Interagency Task Force on Ionizing Radiation

TABLE 2

FEDERAL RADIATION COUNCIL

RADIATION PROTECTION GUIDES FOR GENERAL POPULATION

INDIVIDUALS IN GENERAL POPULATION	500 MREM/YEAR
SUITABLE SAMPLE OF EXPOSED POPULATION NEAR A RADIATION FACILITY	170 MREM/YEAR

TABLE 3

NUCLEAR REGULATORY COMMISSION REGULATIONS

APPENDIX I TO 10 CFR PART 50

NUMERICAL GUIDES FOR DESIGN OBJECTIVES AND LIMITING CONDITIONS FOR OPERATION FOR MEETING ALARA IN EACH LIGHT WATER REACTOR EFFLUENTS (APPLIED ABOVE BACKGROUND)

LIQUID EFFLUENTS	
DOSE TO TOTAL BODY FROM ALL PATHWAYS	3 MREM/YEAR
DOSE TO ANY ORGAN FROM ALL PATHWAYS	10 MREM/YEAR
NOBLE GAS EFFLUENTS (AT SITE BOUNDARY)	
GAMMA DOSE IN AIR	10 MRAD/YEAR
BETA DOSE IN AIR	20 MRAD/YEAR
DOSE TO TOTAL BODY OF AN INDIVIDUAL	5 MREM/YEAR
DOSE TO SKIN OF AN INDIVIDUAL	15 MREM/YEAR
RADIOIODINES AND PARTICULATES	
DOSE TO ANY ORGAN FROM ALL PATHWAYS	15 MREM/YEAR

TABLE 4

ENVIRONMENTAL PROTECTION AGENCY GENERAL ENVIRONMENTAL STANDARD FOR THE URANIUM FUEL CYCLE

40 CFR PART 190

DOSE TO TOTAL BODY OF AN INDIVIDUAL	25 MREM/YEAR
DOSE TO THYROID	75 MREM/YEAR
DOSE TO ANY OTHER ORGAN	25 MREM/YEAR

TOTAL QUANTITY OF RADIOACTIVE MATERIALS TO GENERAL ENVIRONMENT PER GIGAWATT OF ELECTRICAL ENERGY TO BE LESS THAN:

50,000	CURIES OF KRYPTON-85
5	MILLICURIES OF IODINE-129
0.5	MILLICURIES OF PLUTONIUM-239 AND OTHER ALPHA-EMITTING TRANSURANIC RADIONUCLIDES WITH HALF-LIVES GREATER THAN ONE YEAR

TABLE 5

ENVIRONMENTAL PROTECTION AGENCY REGULATIONS FOR RADIONUCLIDES IN DRINKING WATER

MAXIMUM CONTAMINANT LEVELS	
COMBINED RADIUM-226 AND RADIUM-228	5 pCi/LITER
GROSS ALPHA ACTIVITY (EXCLUDING RADON AND URANIUM)	15 pCi/LITER
AVERAGE ANNUAL CONCENTRATION FROM MAN-MADE RADIONUCLIDES (EXCLUDING ALPHA) SHALL NOT PRODUCE DOSE TO TOTAL BODY OR ANY INTERNAL ORGAN GREATER THAN	4 MREM/YEAR

(BASED ON 2 LITER PER DAY WATER INTAKE)

TABLE 6

OTHER EPA AUTHORITIES WHICH IMPACT ON NUCLEAR FACILITIES

- CLEAN AIR ACT AMENDMENTS OF 1977
- FEDERAL WATER POLLUTION CONTROL ACT
  - GUIDANCE TO STATES FOR WATER QUALITY CRITERIA

TABLE 7

ESTIMATED U.S. POPULATION 100 YEAR DOSE COMMITMENT PER 800 MWE-YEAR

Nuclear Fuel Cycle Component	Off-Site Total Body Dose Commitment (Man-Rems)
Mining	110
Milling	39
UF_6 Conversion	6.9
Enrichment	0.022
UO_2 Fuel Fabrication	0.48
Light Water Reactor Effluents	61
Irradiated Fuel Storage	0.0035
Reprocessing	330
Transportation	1.1
Waste Management	8.9
Industry Total	558 Man-Rems or 0.056 Potential Health Effects per 800 MWE Plant per Year

From NRC staff response to Honicker petition

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14. Public Health Considerations of Carbon-14 Discharges from the Light-Water-Cooled Nuclear Power Reactor Industry. Technical Note ORP/TAD-76-3. U.S. Environmental Protection Agency, July, 1976.

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OCCUPATIONAL HEALTH EXPERIENCE IN NUCLEAR POWER

By

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Occupational Health Experience in Nuclear Power*

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Introduction

This symposium is addressing a question of great national concern: "What are the public and occupational health aspects of relying on coal and nuclear power for the generation of electricity?"

This morning's session is devoted to health problems in nuclear power. Our earlier speakers (Dr. Radford and Mr. Harward) have presented information on how radiation exposures are translated into health effects and about the public health effects of nuclear power. I have been asked to discuss occupational health experience in nuclear power from the perspective of a regulator.

In considering occupational health aspects of any fuel cycle, one should address all activities that must take place to obtain the fuel, generate the power, and dispose of any waste materials. In nuclear power, the principal activities are uranium mining, ore processing or milling, fuel fabrication, power generation, and fuel storage and ultimate disposal.

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*Presented at the Symposium on Energy and Human Health: Human Costs of Electric Power Generation, sponsored by the University of Pittsburgh and the Ohio River Basin Energy Study, held in Pittsburgh, Pennsylvania on March 19-21, 1979.

In nuclear power, we can further divide occupational health effects into two categories: radiation hazards and more conventional non-radiological effects. Many of the non-radiological risks of the work place are comparable regardless of the method used for power generation. Examples include scalding, electrocution, exposure to toxic chemicals, falls from ladders or scaffolds, and dropping of heavy loads. Others are not comparable. For example, most of the non-radiological hazards from the mining and transportation of any fuel are basically a function of the tonnage handled and are therefore greater for fossil-fuel than for nuclear energy generation. Exposure to airborne radioactivity in uranium mines has no direct counterpart in fossil power generation, just as black lung in coal miners has no analog in the nuclear area.

Since we in the Nuclear Regulatory Commission are much more concerned with radiological health and because the public interest and mine as well is largely focused on the same area, I plan to spend most of my time this morning discussing occupational radiation exposures to workers in the nuclear field.

10 CFR Part 20 (Reference 1) contains NRC's standards for protection against radiation. Under this rule, whole body doses are limited to 1.25 rem per calendar quarter unless the licensee has determined a worker's lifetime accumulated occupational dose. In this case, the whole body dose is limited to 3 rem per calendar quarter provided that the worker's accumulated dose does not exceed 5(N-18) rems, where "N" is the worker's age (SLIDE 1). You will recognize this as the basic Federal Radiation

Council standard, now under the cognizance of the Environmental Protection Agency (EPA). Last month NRC published (Reference 2) a proposed rule that woule eliminate the 5(N-18) dose averaging formula. The quarterly dose limit would be 3 rem but the annual limit would be 5 rem in all cases.

More important than these limits is the requirement that all doses must be As Low As Reasonably Achievable (ALARA). This basic concept of ALARA is recognized in all radiation standards -- whether they are set by international bodies such as the International Commission on Radiological Protection (ICRP) or U. S. authorities.

In nuclear power plants the ALARA requirement translates into a broad program of actions and controls all directed toward reducing worker exposure. A good ALARA program includes such things as plant design and layout to facilitate maintenance; requirements on fuel cladding, water treatment and purification systems to inhibit corrosion or reduce deposition; controls on access to radiation areas; worker training; radiation surveys; use of remote and semi-remote maintenance equipment; local decontamination of surfaces; special ventilation systems; protective clothing, etc.; etc. -- in short a complex wide-ranging exposure control program.

In fact, ALARA programs are effective in these highly disciplined engineering activities and very few workers receive doses that approach the limits. The next slide (References 3 and 4)(SLIDE 2) presents some recent figures that show where we are. It is obvious that nuclear power plants represent the major source of occupational exposure associated with energy generation -- both in the number of workers affected and in the average dose received. Workers at fuel fabrication and processing facilities also receive significant doses, although they average less than one-third those at nuclear power plants. Perhaps as important as the average dose is the distribution of actual dose received by individuals. This is shown for workers at power reactors and fuel facilities in the next slide (Reference 3)(SLIDE 3). From this one might conclude that there is no cause for concern since of the tens of thousands of workers in the nuclear field only a few hundred approach the exposure limits of NRC's regulations.

However, I believe it is safe to say that most of us here do not accept this view. Recent studies of the incidence of cancer among workers exposed to radiation point to the possibility that the limits may be less conservative than previously thought. While the data are not conclusive on the quantitative effects of low-level radiation, I am personally convinced that in the range of occupational exposures, say from 100 millirem to 5 rem per year, there is no threshold for radiation effects. We do not know the precise relationship between dose and risk at low levels of radiation. However, I believe it is wise that public health officials concluded some years ago that there is a risk, and based exposure standards on assumptions of a linear non-threshold dose response curve. The perception that no radiation dose level is without risk is a key concept underlying ALARA. Therefore, the regulatory requirement that exposures be kept as low as reasonably achievable is of great importance. Simply stated, NRC's aim is to eliminate unnecessary exposure and limit necessary exposures.

The ALARA control program can stress reduction of exposure to individuals or total population exposure (Σ dose x persons exposed) or both. The regulatory approach to achieving ALARA depends on what relationship between exposure and risk is assumed. This sketch, which was prepared by Dr. K. Z. Morgan for recent testimony before a U. S. Senate Subcommittee, may help me to illustrate some of the possibilities. (SLIDE 4)(Reference 5). It illustrates approaches to extrapolating data from observed effects on those exposed at Hiroshima and others who have received high radiation doses, and from limited epidemiological studies, animal studies, studies of the interaction of radiation with body tissues, and observations on behavior of radioactive material within the body. Beginning with data at high radiation levels, the curves show extrapolation down to lower exposures assuming three possible relationships. If the relationship is linear (Curve A), any reduction in total man-rem would be accompanied by a reduction in health effects. If it is sublinear (Curve B), reduction of individual doses at the higher levels would yield health benefits even if this were accompanied by some increase in total man-rem. If, however, the relationship is superlinear (Curve C), the greatest health benefit would be achieved by reducing total man-rem rather than individual doses. And, one would expect for Curve C as well as Curve A, the so-called linear hypothesis, in the case of at least some activities, to see an increase in health effects if the exposure limits were lowered since this could result in more individuals being exposed and, as a result of expected inefficiencies, more total man-rem. This would likely be the case in some nuclear power plant activities such as maintenance and inservice inspection; whereas one would expect little or no increase in total worker dose in the field of nuclear medicine.

In effect, the NRC's regulatory stragety reflects an assumption of a linear or superlinear relationship. That is we are encouraging the reduction of both individual doses and total man-rem.

From the data I showed earlier (SLIDE 2), it is obvious that the greatest room for improvement is in nuclear power plant operation since it accounts for more than 90% of the total dose in the nuclear power cycle. Also, the average exposure is three times that in any other stage of the cycle. As more reactors have come on line, the total number of man-rem has increased rapidly - from 14,000 in 1973 to 21, 000 in 1975, and over 32,000 in 1977, the latest year for which data have been analyzed.

The exposure data can be expressed in man-rem per megawatt year (SLIDE 5)(data from Reference 3). This has been relatively constant with a small increase in exposure at boiling water reactors (BWRs) being balanced by a decrease at pressurized water reactors (PWRs).

The radioactivity in nuclear power plants is initially confined to the core - that is, within the fuel elements housed inside a shielded pressure vessel. With time two things occur. First, the intense neutron radiation in the core converts atoms of material in the structure and in the coolant to radioactive species. A certain fraction of both these is carried outside the pressure vessel in the coolant to other parts of the reactor system where they tend to be deposited on whatever surfaces are present. Second, some percentage of the fuel elements will develop small leaks with time, and some of the radioactive fuel and fission product atoms in these fuel elements will also find their way into the coolant stream and will be deposited on the reactor system surfaces. In general, the more surface area exposed to the coolant in any reactor component, the more important a radiation source it will become. Thus steam generators and other heat exchangers that may have thousands of square feet of surface area are particularly likely to contain large amounts of radioactive material. Demineralizer resin beds used to maintain the purity of water in the core also become a major source of radioactivity. These highly radioactive components are a key element in occupational exposure. Close approach by workers to perform maintenance or inspection could result in significant exposure.

All of this was well known and was taken into account by the designers and builders of nuclear power plants. The areas of high radioactivity were shielded; and those areas routinely occupied by plant personnel such as the control room were further shielded and/or were located as far as possible from the major sources of radiation.

The next slide (SLIDE 6)(Reference 3) listing the percentage of personnel dose by work function shows this. Routine operation and refueling were recognized as important by the plant designers and the ALARA approach was generally applied. Therefore, the doses received in performance of those functions have been constant and relatively small.

In the case of routine maintenance (for example, the repairing of instruments and repacking of valves), control of exposures by the design of nuclear power plants and by careful practice proved more difficult. As a result, for several years this activity was the greatest contributor to occupational exposure in nuclear power plants. By the mid-1970's industry and regulators alike recognized the need for action. Consequently, by improving shielding, equipment, and procedures, industry has substantially reduced exposures from routine maintenance.

Exposures from three activities, however, have doubled in the past few years. These are special maintenance (now the greatest contributor to occupational exposure), inservice inspection, and waste processing.

There have been several occurrences at nuclear power plants that required special maintenance activities. For example, when in 1974 cracks in stainless steel pipes were observed at the Dresden nuclear station, the NRC ordered all operating BWRs to be shut down for inspection of similar piping. Cracks were found at several other reactors. The affected piping had to be repaired or replaced. And in each case the workers involved in the inspection and repair received exposures.

Pressurized water reactors have had a history of corrosion-related failures in the steam generators. This has resulted in the need for workers to plug failed tubes and to remove some tubes for study. In addition, this will shortly result in the full replacement of steam generators in at least four units at two plants. Surry Unit 1 is already shut down for this purpose. The extent of these failures appears to have been unanticipated by the designers. They have been one of the factors that caused the NRC to increase the required frequency and extent of inservice inspections; this in turn has contributed to the doubling of exposure from inservice inspection.

I mentioned that at least two utilities are planning to replace steam generators. Although the principal driving force for these replacements is economic, an important consideration is minimizing radiation exposure to workers at those plants. At the Surry plant, for example (SLIDE 7)(Reference 6), steam generator maintenance accounted for more than 60% of all radiation exposure during 1977. The comparable figure was 40% in 1975 and 1976 and only 6% in 1974, which was before the problems had been observed. The total dose related to steam generator maintenance had risen to 1400 man-rem in 1977. The plant's owners estimate that replacement of the steam generators in both reactors at Surry will result in about 4300 man-rem total exposure. If this number is correct and it has the desired effect of eliminating exposures from steam generator maintenance, the payback time for that 4300 man-rem dose will be about 3 years. Doses not suffered after that time will be the "benefit" of the steam generator changeover. The replacement is therefore claimed to be consistent with ALARA principles.

The NRC staff is taking a hard look at all the proposed activities involved in the steam generator replacements to make sure that all possible sources of exposure have been taken into account in the planning and analyses and that the worker dose due to the replacement is in fact ALARA.

The increase in worker exposure from waste management resulted directly from an NRC decision in 1975 to establish ALARA guidelines for effluents released from nuclear power plants. These guidelines were issued as Appendix I to Part 50 of the regulations (Reference 1). In order to meet the Appendix I requirements, nuclear power plant operators have had to retain more radioactive waste within the plant for longer periods; hence the increase in worker exposure.

Although the examples I have given are from our experience with nuclear power plants, NRC's approach to occupational exposure and its use of the ALARA concept applies to all the facilities and activities that we regulate. As the earlier slide indicated, exposures are relatively low at the fuel preparation and fabrication stages and, since we have no active licensed reprocessing facilities in this country at present, there are no exposures associated with that activity. In nuclear power plants, the principal radiation hazard is exposure of the body or parts of the body from an external source. In some fuel cycle activities, a second potential hazard exists to a much greater degree than in nuclear power plants; that is, inhalation or ingestion of radioactive particles into the body by breathing or swallowing fine particles dispersed in the air. This is of particular concern in fuel facilities processing plutonium. In these facilities special precautions are taken, including isolation of the material in glove boxes or similar enclosures, continuous monitoring, and the use of special clothing and respirators.

Another problem that I would like to mention is how to control exposures to workers employed at more than one facility during a quarter. NRC deliberately places the burden of controlling access, monitoring workers, and reporting exposures at a facility on the facility licensee. This, however, does not take into account employees who moonlight on a second job or who may leave one employer and go to work for another within the same quarter. The records of each employer may show that the worker's exposure is within acceptable limits; but the sum of all the exposures may show that the worker has been exposed beyond the limits.

The Commission is now considering issuing a rule that would require licensees to control total occupational dose for workers at their facility. The licensee would be required to get from workers a statement of exposure from outside sources during the quarter. Each terminating worker would be furnished, on request, an estimate of the doses received. The worker could then make this available to his next employer to help ensure that he is not overexposed.

The Department of Health, Education, and Welfare (HEW) recently published the data presented in the next two slides (Reference 7) (SLIDES 8 and 9). These data show that the radiation exposure received by workers and the public from nuclear power is a small fraction of the total received from other sources. Nevertheless, we believe that any exposure may result in some risk and accordingly try to reduce exposure to the lowest levels reasonably achievable.

Occupational exposure to radiation is a health hazard. As with other public health hazards that cannot be completely eliminated, a judgment must be made as to what level of exposure is acceptable. Affected workers must have a basis for informed acceptance, that is, workers should be aware that risks exist and of the extent of those risks when they accept employment at nuclear facilities just as workers at mines, steel mills, chemical plants, and so on should be aware of the different risks facing them in their employment. Open discussion of these risks, including meetings such as this, helps to contribute to the informed judgments that must be made.

I would like to conclude by responding to a question that has been asked several times of late; that is, In light of the current uncertainty regarding the health effects of low-level radiation exposure, why doesn't the NRC reduce the existing permissible radiation dose limits?

It is important to understand that dose levels such as the 5 rem per year standard for workers, as used in regulatory practice, are upper limits of permissible doses to individuals which, if exceeded, set off a chain of regulatory actions such as investigations, reports, mandated corrective actions, and possibly penalties. These levels should not be taken as acceptable or safe in themselves; that is, a person is not in immediate danger if he receives slightly more than the permissible level, nor is he perfectly safe from adverse effects if he receives slightly less than the permissible level.

Radiation dose standards, as applied by the NRC, are more correctly characterized as a broad range of radiation control measures intended to assure that actual doses received by individuals remain far below the prescribed permissible dose limits. Experience has shown that these control measures have been effective in keeping occupational exposures well below the limits.

Even so, we have noted that the total collective dose to workers in NRC-licensed activities has been steadily increasing during the past few years. Further, we are aware of recent health studies that raise a question as to whether current estimates of health effects are as conservative as previously assumed. Because of these two points, we have initiated several actions aimed at tightening up our radiation standards to further reduce the collective dose.

We have proposed a rule change that would eliminate the special provision that currently allows some workers to receive as much as 12 rem per year. We are in the final stages of a rule change that would provide more effective control over doses received by workers who move from one licensed facility to another and those who work at more than one licensed facility at the same time. We are considering a rule change that would strengthen our commitment to the "As Low As Reasonably Achievable" (ALARA) concept for occupational exposures.

The NRC staff is also in the process of issuing a series of regulatory guides to describe acceptable ALARA techniques for different types of licensed activities and to help achieve better radiation exposure control.

We believe that it is these types of regulatory standards actions that will be most meaningful, in a practical sense, in reducing radiation exposures. Lowering of the existing permissible dose limits is also under consideration. However, by itself, such an action may not be as effective as the tightening of radiation control measures discussed above. In fact, for some regulated activities lowering the limits might be counterproductive, that is, it might increase the collective dose.

With respect to the permissible dose levels, we are currently cooperating with EPA in its development of Federal guidance on occupational exposures. We anticipate joint participation with EPA and OSHA in a public hearing to be held later this year on the adequacy of the present standards.

In summary, we are taking action to tighten up our regulatory requirements to further reduce occupational exposures. We have not concluded that the evidence presently available indicates an immediate need to lower the present permissible dose levels; but we are, along with EPA and OSHA, examining the adequacy of these present standards.

REFERENCES

1. Code of Federal Regulations, Title 10 - Energy, Chapter I - Nuclear Regulatory Commission, revised as of January 1, 1978, Washington, D. C., U. S. Government Printing Office (1978).

2. 44 FR 10388, Federal Register, Volume 44, No. 35, February 20, 1979.

3. "Occupational Radiation Exposure, Tenth Annual Report, 1977, NUREG-0463, U. S. Nuclear Regulatory Commission (1978).

4. EPA data from report to be published.

5. K. Z. Morgan, "Radiation Induced Cancer in Man", presented to the Subcommittee on Energy, Nuclear Proliferation and Federal Services, U. S. Senate, March 6, 1979.

6. Data compiled by NRC staff.

7. Summary of Work Group Reports of the Interagency Task Force on Ionizing Radiation, Department of Health, Education and Welfare, February 27, 1979.

SLIDE 1

NRC REGULATIONS LIMITING RADIATION DOSE

1.25 REM/CALENDAR QUARTER

OR

3 REM/CALENDAR QUARTER

IF ACCUMULATED DOSE IS KNOWN TO BE

$< 5(N-18)$ REM

(N = WORKER'S AGE)

SLIDE 2

RADIATION EXPOSURE OF U.S. NUCLEAR FUEL CYCLE WORKERS

ACTIVITY	NUMBER OF WORKERS		MEAN WHOLE BODY DOSE FOR THOSE EXPOSED (REM)	COLLECTIVE DOSE, MAN-REM
	TOTAL	EXPOSED		
URANIUM MILLS[2]	300	100	0.050	5
URANIUM ENRICHMENT[2]	7,471	5,664	0.070	400
FUEL FABRICATION AND PROCESSING[1]	11,496	7,004	0.246	1,725
NUCLEAR POWER PLANTS[1]	71,904	44,233	0.740	32,731

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[1] NRC LICENSEE DATA FOR 1977.

[2] EPA ESTIMATES FOR 1975.

SLIDE 3

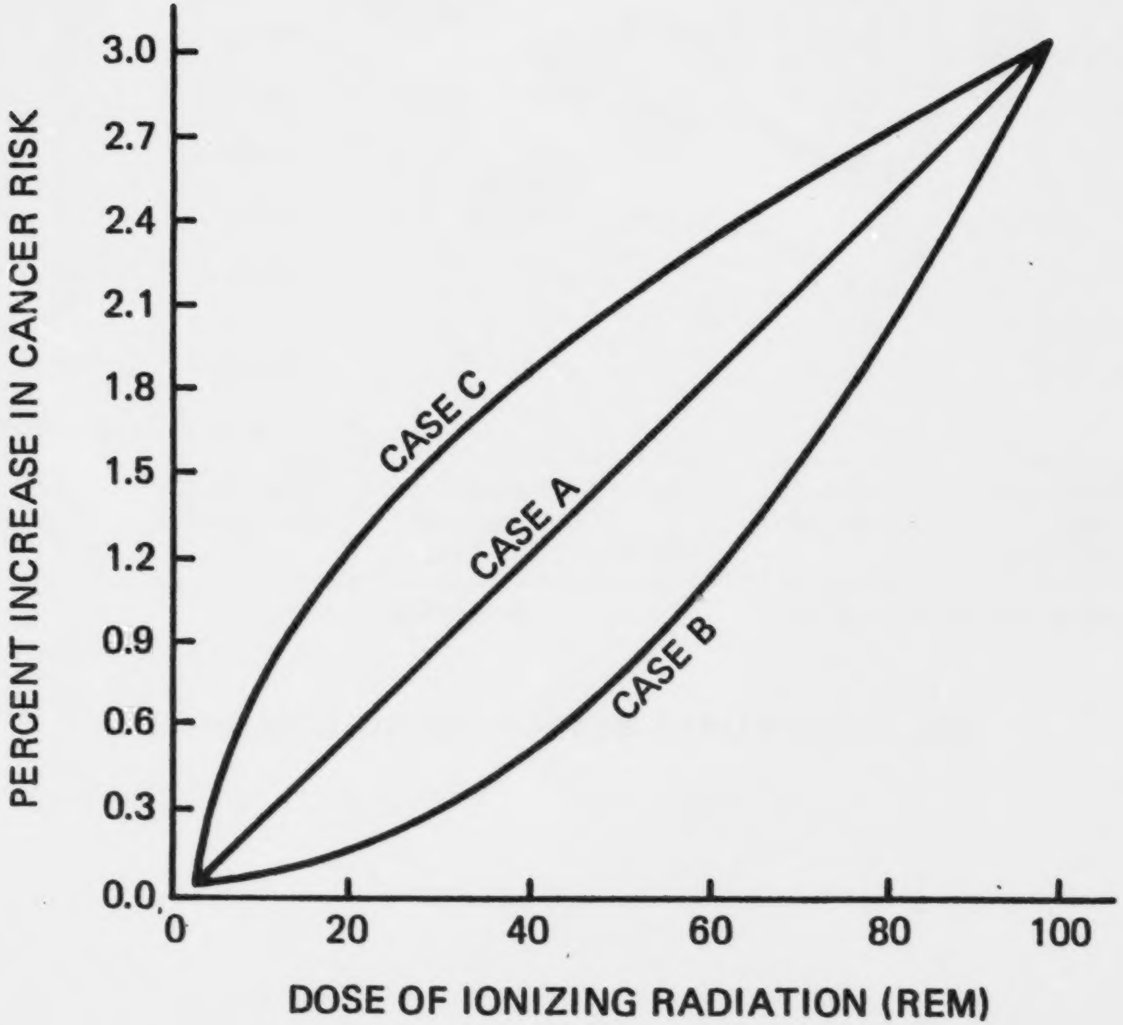
DISTRIBUTION OF WORKER EXPOSURE IN 1977

DOSE RANGE REM	POWER REACTORS		FUEL FABRICATION AND PROCESSING	
	NUMBER OF WORKERS	PERCENT OF THOSE RECEIVING MEASURABLE DOSE	NUMBER OF WORKERS	PERCENT OF THOSE RECEIVING MEASURABLE DOSE
NOT MEASURABLE	27,671	–	4,492	–
MEASURABLE BUT < 0.10	15,523	35.0	4,533	65.0
0.10 - 0.25	6,750	15.0	1,057	15.0
0.25 - 0.50	5,179	12.0	571	8.0
0.50 - 0.75	3,300	7.0	315	4.0
0.75 - 1.0	2,500	6.0	179	3.0
1 - 2	6,174	14.0	205	3.0
2 - 3	2,838	6.0	91	1.0
3 - 4	1,130	3.0	28	0.4
4 - 5	569	1.0	25	0.4
> 5	270	0.6	0	0.0

PERCENTS DO NOT ADD TO 100 DUE TO ROUNDING.

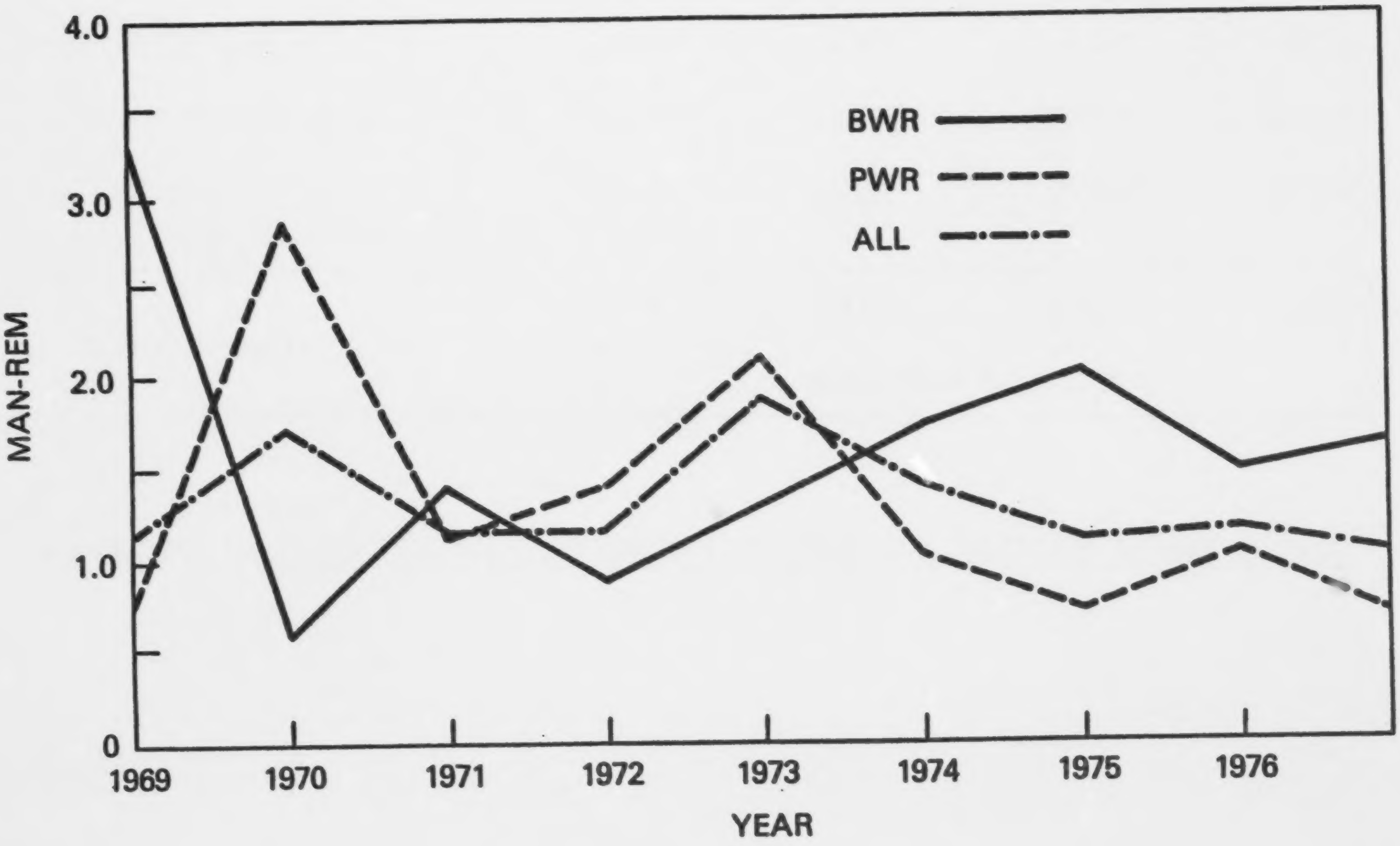
SLIDE 4

CANCER INDUCTION AS A FUNCTION OF DOSE OF IONIZING RADIATION FROM 0 TO 100 REM



SLIDE 5

AVERAGE MAN-REM/MW-YR



SLIDE 6

PERCENTAGES OF PERSONNEL DOSE BY WORK FUNCTION

WORK FUNCTION	PERCENT OF DOSE			
	1974	1975	1976	1977
REACTOR OPERATIONS AND SURVEILLANCE	14.0%	10.8%	10.2%	10.6%
ROUTINE MAINTENANCE	45.4%	52.6%	31.0%	28.9%
IN-SERVICE INSPECTION	2.7%	3.0%	6.0%	6.6%
SPECIAL MAINTENANCE	20.4%	19.0%	40.0%	41.4%
WASTE PROCESSING	3.5%	6.9%	5.0%	5.9%
REFUELING	14.0%	7.7%	7.9%	6.6%

SLIDE 7

SURRY IN-PLANT DOSES

ALL OPERATIONS

YEAR	TOTAL INDIVIDUALS EXPOSED	DOSES (MAN-REM)	AVERAGE REM/INDIVIDUAL
1974	1,715	844	0.5
1975	1,948	1,649	0.8
1976	2,753	3,164	1.2
1977	1,860	2,307	1.2

STEAM GENERATOR MAINTENANCE

YEAR	TOTAL INDIVIDUALS EXPOSED	DOSES (MAN-REM)	AVERAGE REM/INDIVIDUAL	TUBES PLUGGED
1974	26	54	2.1	200
1975	393	638	1.6	500
1976	711	1,287	1.8	1,750
1977	–	1,410	–	1,525

REPLACEMENT OF STEAM GENERATORS 4,336 (EST.) MAN-REM.

U.S. GENERAL POPULATION EXPOSURE ESTIMATES — 1978

SOURCE	PERSON-REMS PER YEAR (IN THOUSANDS)
NATURAL BACKGROUND	20,000
TECHNOLOGICALLY ENHANCED	1,000
HEALING ARTS	17,000
NUCLEAR WEAPONS	
FALLOUT	1,000 — 1,600
WEAPONS DEVELOPMENT, TESTING, AND PRODUCTION	0.165
NUCLEAR ENERGY	56
CONSUMER PRODUCTS	6

U.S. OCCUPATIONAL EXPOSURE ESTIMATES — 1975

SOURCE	PERSON-REMS PER YEAR (IN THOUSANDS)
HEALING ARTS	40 -- 80
MANUFACTURING AND INDUSTRIAL	50
NUCLEAR ENERGY	50
RESEARCH	12
NAVAL REACTORS	8
NUCLEAR WEAPONS DEVELOPMENT AND PRODUCTION	0.8
OTHER OCCUPATIONS	50

DISCUSSION

SESSION V

Jerry Rosen, University of Pittsburgh: I have two brief questions. Dr. Radford, you indicated in your talk that the incidence of cancer from natural background would be in the order of one or three percent and Mr. Harward indicated, on his first slide, that about 1/2% of the cancers in the population would be due to natural background. I'd like you to address that. Another item for any of the panel members would be that on many of the graphs we've seen here today we see error bars on measured health effects; for example, incidence of leukemia. We very rarely see error bars associated with the delivered doses. Could you indicate what order of magnitude these errors might be?

Dr. Radford: I'll take the first part and possibly Dave Harward might want to comment. The estimates I gave are based on more recent data than the 1972 data. In part, these data are now published in the 1977 UNSCEAR Report which is in the open literature. I think they indicate the impact of the "further follow-up effect," if I can call it that. One of the big problems in defining the cancer risk from radiation exposure has been, as I tried to indicate in my presentation, how long is the effect going to last within the population. If it lasts throughout the lifetime of the individuals, it means that the risks are substantially higher than if, as in the case of leukemia, it doesn't last for the lifetime of the individual. I think that previous risk estimates have, to a considerable extent, made the assumption that the risk will last only for a finite period of years and now we think that most cancers will not. Mr. Harward, do you want to comment on that?

Dave Harward: I think the table you're referring to is the one that I got out of the HEW Draft Interagency Report, where it indicated something like roughly 50% of the radiation induced cancers in the United States, might be due to natural background. As to your comment on the error band being rather sizable, I think this is generally the case with this kind of thing and error bands with a factor of two when trying to interpret some of these numbers is certainly not uncommon.

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Dr. Radford: Just to correct you, Dave. You meant a 1/2%, not 50%.

Dave Harward: 1/2% of the total cancer deaths, but about 50% of the total radiation induced deaths. If you look at the HEW figures with about 4,000 theoretically radiation induced cancers per year, roughly 2,000 are from natural background.

Dr. Radford: Can I just respond to the dose issue. The speaker, Mr. Rosen, is quite correct that one of the problems that we have in the epidemiology of almost any environmental agent, is to define what the exposure has actually been. Now, the dose data that I used for the Japanese is based on the most recent tissue doses obtained by the Oakridge National Laboratory. For the low dose end of the curve I think the error bars are relatively small, becauses it was much more possible to define the shielding characteristics of the individuals who are out away from the bomb epicenter, in contrast to the higher doses where I think the uncertainties remain somewhat high. So I agree with your general point and maybe we should, if we had the information, put error bars on the doses.

Mr. R. Minogue: I'd like to add to what Dr. Radford has said, looking at a different exposed group. First of all, I think there is always a difficulty in trying to relate some of the epidemiological studies of exposed workers, or some of the studies planned on soldiers, to the medical exposure that the people studied may have received. This is a matter of some real concern.

Even as to the radiation exposure involved in the activities of these various groups, there's a lot of uncertainty. For the soldiers and other personnel involved in the weapons test program, the external dosimetry was at best limited and the internal dosimetry verged on nonexistent. Thus there's a great deal of uncertainty as to what the exposures really were, at least in my opinion.

Even among the industrial radiation workers that are involved in highly disciplined activities where the ALARA concept has been applied, there is a great deal of uncertainty as to the doses received. Let me just tick off some of the key points. First, the low exposed part of the group you almost have to drop from your data base because among them may be many workers who received no exposure or some indeterminate low exposure. Below about 30 millirem on the film badge, the data verges on indeterminate. Even above that level there's a great deal of variation in the data that one gets back if you use one of the commercial dosimeter processing services, in terms of the doses reported. This is a matter that we're giving a great deal of attention to right now in the context of an epidemiology feasibilty planning study which we've been mandated by Congress to do. I think it may be a statistical variation, but it's fairly wide. The point I'm trying to make is there is, in fact, a fairly large plus or minus on the external exposures measured by the film badges and other personnel dosimeters used in the industry.

The last point I'd like to make, in brief, relates to internal dosimetry. That type of exposure is quite limited in the regulated industry because it's generally been regarded as somewhat of a "no no" because of the recognized high level of uncertainty. There are a number of techniques that are applied, bioassay techniques, for example, for workers who may have been exposed to internal doses that I think are quite good. The dosimetry, in fact, is probably rather good between measurements of air sampling systems and bioassay.

Dr. Wald: We'll take somebody from the front. While he's getting there, let me mention that the Ichiban program, the Japanese reconstructed dosimetry program, felt that they had arrived at something that was approximately plus or minus 25 to 50% of each individual's exposure. The other point I think has to be recognized is that the medical exposure risk was as great for people who received insignificant weapons radiation in Hiroshima and Nagasaki because they all came under an A-bomb Survivors Compensation Law which provided for medical examinations, so the lower the exposure from the weapon, the proportionately greater influence the medical x-ray in the following would have been.

Dr. Radford: If I may, Neil, I just want to point out that very careful assessment of what the medical X-ray contribution has been to various tissues-- and it's by no means uniform--has shown it's not the whole body approach that has been taken into account. I'm not sure that all the data I showed has built that in, but I think we can .

Dr. Wald: Yes. Usually it just shows the weapons exposure, but I think that's a good point.

Dr. H. Spencer, one of the ORBES researchers: I am interested particularly in the Ohio River Basin. In the basin we consume something on the order of 400 million tons of coal a year. Coals in the basin contain one to two parts per million uranium and thorium oxides, in equilibrium with their decay daughters. Upon combustion the gaseous components, thoron and radon, go out immediately. Some of the other daughters apparently evaporate but the uranium and thorium appear to remain with the ash. The question I'd like to direct to the panel can be answered yes or no. Should we now begin looking at these ash pits the same way we're looking at mine tailing areas across the country?

Mr. Harward: I can take a stab at what I know is going on about the subject. I mentioned earlier the EPA implementation of the Clean Air Act Amendments of 1977. They are looking at this very closely through a series of contracts and have a substantial background of data over roughly 10 years from fossil fuel plants. I do know they are considering whether or not to include fossil plant radioactive emissions within the Clean Air Act. By August of 1979, they have to construct some lists of various pollutants that would be covered under the Clean Air Act. I won't go into what they are, but there are three sections in the Clean Air Act under which pollutants can be listed. This is under consideration. I don't know at what point they are in their work on it.

Dr. H. Spencer: I would like the NRC representative to answer the question.

Mr. Minogue: I really don't feel that falls within the range of my regulatory expertise. I think Mr. Harward is better qualified to answer that.

Dr. Wald: I think we have to allow yes, no or none of the above.

Dr. J. Hartnett, University of Illinois, Chicago: I have two questions that are directed to the last two speakers. I was somewhat confused by what I thought were different statements. I thought I heard the second speaker, Mr. Harward, say that the uranium mining and milling pose greater hazards with respect to radiation than did the nuclear power plant. And I thought I heard the last speaker, Mr. Minogue, say just the opposite. I'd like to have that question clarified. The second question is directed to the last speaker, Mr. Minogue. I believe on the last slide, if I read it right, it said that other occupations show 50,000 person-rems per year, while nuclear energy showed 50, nuclear reactors 8, and nuclear weapons 0.8. Does this suggest that a worker in a candy factory or a professor in a classroom is exposed to more radiation than a worker in the nuclear industry or in the nuclear weapons industry?

Mr. Minogue: Let me answer the second question first because it is pretty straightforward. What it suggests is that there are a number of people, far larger numbers of people, in some of these other occupations who are not generally regarded as radiation workers but who are exposed in the work place to radiation at levels greater than that which arise from natural background. Because there are so many of these, and if you recognize the no-threshold concept, the total number of man-rem they receive is quite significant. This would include phosphate workers, it would include the airline stewardesses and so on. There are large numbers of these people.

Dr. J. Hartnett: You did not mean by that "all other occupations"-- you are talking about selected occupations. Is that correct?

Mr. Minogue: The data I'm referring to is from the HEW Report. My understanding of the groups that they looked at was that they were all workers who are exposed to radiation at higher than background levels because of the nature of their occupation. There are many of these workers. The data is not terribly good, but there are very large numbers of these workers.

Dr. J. Hartnett: You clarified that slide, but I think as it's stated on the slide it is misleading because it says "other occupations."

Mr. Minogue: Let me now reply to part of the first question. Certainly I intended in no way to imply that the public exposure problem from mill tailings is trivial. And I'm sorry if you read that into what I said. My topic was occupational exposure and the occupational exposure to radiation of mill workers is a very minor problem compared to the occupational exposure to nuclear power plant workers. First, there is a large number of those people in the nuclear power plants; and secondly, they are inherently exposed to very high radiation levels. The work they do in which they receive exposure is primarily related to maintenance of critical safety equipment; nonessential exposures are rather easily eliminated. They are, as a group, exposed to quite high radiation levels. I associate with them; I've been exposed to those radiation levels myself, and they're quite high.

Mr. Harward: Well, just to further amplify. The table that I had on the screen was for environmental exposure and according to the NRC data, the combined mining and milling population dose represents a little better than a 1/3rd of the total dose from the entire nuclear fuel cycle. The only thing that was higher was the fuel reprocessing public exposure which I indicated, of course, is nonexistant at present, because of the lack of fuel reprocessing.

Mr. Olson, Westinghouse: I have two questions I'd like to address to Dr. Radford. First of all, I'm surprised at your definition of low level radiation. As I think I understood you, you said from 10 to 100 rads. Just a minute ago, though, Mr. Minogue was talking about high levels of radiation, he was talking about five to fifteen. So my question is what about levels less than, say, ten rad. What are the effects there?

Secondly, at these exposures of ten to one hundred rads, is there any difference in the rate of exposure? That is, is it the same getting a 50 rad dose in a few seconds, or a few minutes, as it is getting a fifty rad dose spread over a year?

Dr. Radford: Right, good questions. Really, the second question leads into the first. The question of dose rate has been one that has been of considerable interest and importance in the radiation protection field. In other words, the point you raised, if you get the radiation stretched out over time, let's say accumulate 50 rads that way, compared with getting zapped with 50 rads all at once, does it make any difference in the long term effect? For the kinds of radiation represented by alpha radiation, the human evidence suggests that stretching out the dose increases the risk per total accumulated dose. That's for so-called high LET radiation; that is, radiation with a high linear energy transfer. For low LET radiation, the experimental data strongly suggests that stretching out the dose reduces the risk per rad per total accumulated unit of dose. And until comparatively recently this has been rather the accepted view in interpreting risks of relatively low doses. If we use the linear hypothesis, and if we take no account of the so-called "dose rate effect" we will be overestimating the risk for low LET radiation, and possibly overestimating it by a significant amount. I think follow-up of the occupational groups which has now been stimulated by a number of these recent studies is going to help us out a lot on this. We have some evidence which suggests that the dose rate effect for even low LET radiation is not as significant as we thought it was, based on the animal data, and there are theoretical reasons why the animal data might have shown this more readily than the human data. Briefly, these are the comparisons of the cancer risk arising in the women who were radiated by fluoroscopy repeatedly over many months or years, turn out to be almost identical with the risks per unit dose that were obtained from women who were treated for other conditions and received a single dose of radiation, in the order of 100 rads or so, to the breast tissue, compared with the Japanese A-bomb survivor data when again it was a single dose at relatively high doses in this case, too. So, the previously thought to be applicable "dose rate effect for low LET radiation" is not strongly supported by the human data at this time. In either case, both for the high LET radiation which leads to an underestimate of the risk at low dose rates, let's say, and the low LET radiation which might lead to an overestimate of the risk, the uncertainty probably is not very much greater than a factor of two. So, if you'll accept that degree of uncertainty, which I think we have to because of the data, then the dose rate effect becomes relatively minor.

Dr. N. Wald: Are you specifically addressing the endpoint of cancer induction?

Dr. Radford: Yes, strictly cancer induction. Obviously for other endpoints such as the kind that Dr. Wald is particularly interested in, dose rate makes a big difference.

Mr. Minogue: I'd like to make a point in this connection. I appreciate the questioner raising this. I noted the same difference in point of view when I listened to Dr. Radford's speech and meant to comment on it in giving my own remarks, and then neglected to. He and I don't cut off at the same point. When I talked about occupational levels or low-level exposure, recognizing what the averages are and recognizing the turnover in job assignments and so on, I am definitely thinking of a range that cuts off from 10 to 20 rem lifetime exposure. And it's clear that Dr. Radford is looking at a much broader span, that is much higher lifetime exposures.

Dr. E. Radford: Well, I'd like to comment on that. I'm sorry I didn't answer that question because I think it's an important one. If the dose rate effect is minor, as I have just indicated, then it doesn't matter. What we're talking about is total cumulative dose up to particular age or time. And there is I think, for example, from background radiation exposure up to age 50, you've accumulated five rads. Well, you're already at five and you're going to add to that from your medical exposure another four or five rads. So you're up to ten already, and now we're going above that with these other exposures. So, with regard to the general population, yes, we are concerned in the range of an extra added dose of, maybe over the lifetime of an individual, a few rads. I agree that in the epidemiologic data literature, we don't have a lot of hard data at those doses, and therefore we are extrapolating downward using one of these models in order to get an indication of what effect there will be, say from 10 to 20 to 30, to 50 rads, where we do have some data and where the data are actually becoming quite good.

Now with regard to Mr. Minogue's statement about the occupational exposures, this gives me an opportunity basically to ask the other panelists a question. They used aggregated data. In other words, they averaged over the industries. I would like to have them comment on the fact, and it's my reading of the NRC reports, that the performance of different reactors within the same type of design and everything else, is widely variable; I mean by a factor of almost 10. So that some plants are restricting their exposures to workers very markedly and indeed are achieving what I consider to be an admirable record, whereas other plants are not. When you average them all out, obviously then, you set an intermediate value. The second thing is that if you look at the aggregated data in the UNSCEAR Report last year (1977), worldwide, but even in the United States, you find that there are substantial subsets of the occupational groups that have now already accumulated 50 to 60 to 70 rads, and presumably they are going to continue working. There are some of the foreign plants that aren't doing as well. They are up over 100 rads to individual workers and I don't mean only one worker--I mean a few, at least perhaps 1% of the total work force. So when we talk about occupational exposures, we're not talking about five rads or ten rads. We're talking about 50 rads or 100 rads over the work life of the individual. You disagree, Bob?

Mr. Minogue: Let me take the last point first. Yes, I definitely disagree. There may be a few individuals who receive those levels but that's simply not true of the vast majority of the workers. The other comment you made is, I think, an important one. There are differences among plants. Some of these are clearly due to different operating experiences; some have had more problems than others. The older plants have had some build-up of activity. Some plants have had more modifications imposed by the regulators than others. And many of the differences can be attributed to this. But there are differences that relate to the -- let us call it -- "the level of conviction" with which ALARA is applied, and because of this the staff of NRC has recommended to the Commission that we find a regulatory framework to put more teeth in ALARA. Not from a perception that many of the companies haven't done a very good job, because they have, but to get hold of the people who aren't measuring up and bring them into line as well. That element is clearly there. It isn't all attributable to differences in the operating experience or maintenance requirements for the plants.

Tom Zeller, Indiana State University: I have two questions for Dr. Radford. I walked in a little late on your talk, so if you addressed these, I apologize. First of all, your figure of half a percent of cancers, I hear you mentioning half the percent of the cancers are due to the background radiation, is that what I was picking up?

Dr. Radford: No, I said one to three percent.

Tom Zeller: One to three--I'm wondering how you could ever get that number without just plain guessing. Secondly, I'm really interested where you got that number. Dr. Goffman's study, which is about ten years old now, estimates that the total United States population is exposed to five millirem dose--you'd end up with thirty-two thousand excess cancers per year or excess deaths, I don't remember which. Now you're saying that this is three orders of magnitude too large a figure?

Dr. Radford: I'm not sure where the three orders of magnitude came from.

Tom Zeller: You're saying a thousand or two thousand at background, which is two millirems, and he's talking five millirems.

Dr. Radford: Five millirems he calculated? No, I don't think so. I worked with Dr. Goffman closely at various times when he was generating those numbers and I don't think that's correct. We can come back to that point. I would prefer simply to say that the estimate that I derived is based on the linear hypothesis, and I was careful to say it. In other words, taking the data we have at the higher doses and extrapolating them downward to what the individual gets over his life span from background radiation, leads to one to three percent of the cancers. In terms of incidence that's around 600,000 cancers a year, so 1% to 3% is 6,000 to 18,000 that would be due to background radiation.

Tom Zeller: When Dr. Goffman was generating those numbers, I thought it was with regard to the acceptable radiation limit around the fence of the plant. Isn't that five millirem?

Dr. Radford: No, that's five hundred millirem.

Tom Zeller: Okay, so was that the figure he was using?

Dr. Radford: I believe so.

Tom Zeller: Is that figure in the ball park with what he comes up with? I've heard that figure disputed, is why I ask.

Dr. Radford: I think his estimates were too high and the principal reason was that he made the assumption that all cancers induced by radiation had the same sensitivity; that the percent increase in risk of cancer of any type was proportional to the dose and was equal for all different cancers. I think that assumption has been very thoroughly contradicted by the human data.

Tom Zeller: My second question is--and maybe you mentioned this also--I didn't hear anything about the recent Mancuso study and the study of the Naval Shipyard workers that involved, in both cases, very low dosages. It's been in the media, but I don't have the data on what they were exposed to or how significant were the number of cancers. Do you have some information on that? Thank you.

Dr. Radford: Okay, I'll take that one, and I'll let the other panelists add any comments they would like. I've discussed this with Dr. Mancuso so this is, in no way, telling a tale out of our school. In my opinion, the Mancuso study shows a relationship of risk of cancer to radiation exposure in the work place, at the levels at which those workers were exposed, but only for one cancer type, namely multiple myeloma, which is a relatively rare, although rapidly increasing in importance, type of bone marrow cancer.

. The other putative effect, an increase in pancreatic cancer in these workers, I do not believe can be adequately ascribed to the radiation exposure. I think it's a linked effect and it's a problem in our methodology of epidemiology that we frequently cannot assign a single cause or even a direct cause to a particular agent.

If you take the excess risk that these workers had, and I agree that there was a small excess risk, and compare it to the expected risk on the linear hypothesis that would be derived from the figures that I used, it comes out very close to what you would have expected. Indeed, you would not have expected all to be in multiple myeloma, but you would have expected a scattering of cancers, but unfortunately the epidemiologic technique is just not capable of detecting it.

So my first conclusion is that the Hanford data did not show any unusually high risk in these workers. The second point, and I believe Dr. Mancuso agrees with me on this, and I have repeatedly said this in public, my interpretation of the Hanford Study is that the exposures were remarkably good, remarkably low. Here you had a plant that started out making plutonium for military purposes way back in the forties and went through the whole range of nuclear technology including peaceful uses and military uses and everything else, starting from absolute scratch with relatively untrained personnel, and yet, when you look at the exposure data in that plant, it is extremely good. The average dose over the working span of the individuals, many of which were short, I agree, because they were construction workers and they left, was only 1.8 rads. And Alice Stewart herself has commented to me her surprise that so few workers ever got more than five rads a year. Very few ever did. So my conclusion is that this plant was extremely well run from the health physics point of view, and I have had occasion to congratulate Mr. Herb Parker who was the chief health physicist at that plant for this fact. And indeed if anybody were here from the union, I would tell them you've got a well-run plant. All you have to make sure of is that it keeps being well run. So with regard to the Hanford Study that's my bottom line and maybe some of the other panelists would want to comment on this.

The second point is the Portsmouth Navy Yard Study. Drs. Najarian and Colson were very careful in their paper, published in Lancet, to point out that it was a preliminary study; they had no dosimetry. There is no possible way you can compare, let's say, the risks that we would derive from the other studies where we do have dosimetry. They are now in the process of collecting the dosimetry data and bringing together the data on mortality. I hope that it will be possible for NIOSH and other groups that are involved in this study, to blend their data together. So the Portsmouth Yard Study in no way says anything about whether the risk is higher, lower or what.

The same thing can be said for the Utah children who have recently been studied from, and whose exposure to radiation was from fallout from bomb testing. Again, no dosimetry is adequate and it is premature to say anything about that result except for one important thing. And that is they have found an excess of leukemia in the children. This result suggests that the dose response curve in children might conceivably be different in shape from the dose response curve in adults. As you noticed, I used the curvilinear dose response curve for the adults, primarily adults in the Japanese data.

So I conclude that of all the, "recent studies that show an increased risk" none of them really do. The only one is Dr. Bross's reanalysis of the Tri-City Study of children irradiated in utero. I simply do not agree with Dr. Bross that his data shows what he says it shows.

Dr. Wald: Do any of the panelists want to respond?

Panelist: I'd like to make a comment about the first question from the gentleman from Indiana State. I am familiar somewhat with Dr. Goffman's projections. These were made approximately ten years ago. And I think the key thing to keep in mind is that those projections, of total cancer deaths, were based on two hundred million people in the United States, each being exposed to the five hundred millirem per year. If you look at the actual exposures of the public, and I presented these in another format in one of my slides, you'll find that the exposure, per capita, in the United States historically has been well less than 1 millirem per year which is akin to riding an elevator maybe ten times a year in terms of the change in background. I think you've got to keep in mind these projections of a lot of people to doses which can get astronomically big, but it has to be kept in perspective.

Dr. Wald: I think the fatalities from the crowding at the fence post, if you put the whole United States population in, will be much higher than anything Dr. Goffman contemplated.

Mr. Minogue: I'd like to comment on the work of Drs. Mancuso, Bross and Alice Stewart, and others, from a little different perspective from that of Dr. Radford. I'm a regulator and I have to make some immediate decisions with the industry that I'm regulating now. As these studies began to come out, certainly from the beginning I recognized the kind of methodological challenge that has been raised to some of the conclusions. Dr. Radford has identified a number of them here, and the statisticians, from a mathematical point of view, have raised equally sharp challenges. However, the fact does remain that these studies would indicate, and I referred to this twice in my remarks, that the basis of the present radiation protection standards perhaps is not quite as conservative as some of the regulators thought it was. In other words, let's say it gives a lot more emphasis to the importance of applying the linear hypothesis literally as if you really mean it. So I would say as far as what we on the NRC staff have done, between the trend analysis of exposures that I covered in my remarks in some detail, and looking at these data and considering discussions we've had, particularly with Drs. Bross and Alice Stewart--that these were factors in leading us to conclude that, even though there may still be uncertainty and there certainly is not widespread scientific acceptance of the findings of these researchers, there were enough indications here that we as regulators should look hard to redoubling some of our efforts to control exposure, which is what we're doing.

John Blair, Evansville, Indiana: Concerning day workers in nuclear plants and how that practice is going today where a worker can come in and receive his yearly dose of radiation one day and then not work in the plant any more. Just what is happening in that level?

Dr. Radford: I did address the genetic effects of radiation, and I do think they are important. You were apparently not here when I did address it. I think my position on the genetic effects is that you cannot really add them directly to the somatic cancer effects, the way the ICRP has done, for example. They equate "health effects from genetic damage" with the "health effect from cancer induction." This a real problem and I know the geneticists share this concern. That does not mean that the genetic effects are ignored, far from it. I think what it tells me is that we must be especially conservative in restricting exposures, especially to individuals who will be in the child-bearing age or up to the time of the usual reproductive span of the individual. One way to interpret this is to restrict very sharply the exposures of young individuals working in association with the radiation, whether it's in the nuclear industry or whether it's in general industry as we've heard about, or whether it's in medical uses, restricting exposures of individuals who are under the age of 35 as sharply as you can. I think there is going to be undoubtedly a lot of discussion in this area as Mr. Minogue's review gets under way. So I don't mean to play down the genetic effects; I think they're very real and, as I try to indicate quantitatively, they look as though they are in the same ball park in terms of percent effect. But you just can't compare a genetic change directly with a cancer change.

John Blair: Have there been studies done about the genetic effects to date that adequately addressed the problem, in your view?

Dr. Radford: The human studies are plagued by the fact that we don't know what the natural genetic load is in the human population to begin with. So if you don't know what the "normal level" is, which we know very accurately for cancer, you see, then if you look at a population like the Japanese bomb survivors or any other population of human subjects, it's almost impossible to detect whether environmental agent A, B, C or D has been having an effect of a genetic nature, in part, because it may be expressed only in two or three generations away and that's 60, 80 years from now. So we're very seriously limited by that problem. The spiderwort: I'm familiar with Dr. Ishikawa's work. The studies of plant cells have shown that you get detectable genetic effects down in the range of a few rads. And it's one of the experimental pieces of evidence that convinces us that the "linear no-threshold dose response curve" is not just a mathematical convenience but it has some substance in the biological sense. And it's not the only data, by any means.

Dr. Wald: Bob, do you want to take the other question?

Mr. Minogue: I'd like to comment on the genetic question. I didn't mention it in my talk because Dr. Radford had covered it. I wouldn't want to leave anybody with the impression that we, as regulators, discount the genetic problem. There is a tendency, I think, because of the relatively easier experimental problem that the researchers in this area tend to talk mostly about cancers, but the regulators recognize that there are other adverse effects. In fact, I think it's true that some of the decision makers among my predecessors back in the 50's were more concerned with genetic effects in considering how to treat low levels of exposure. One of the reasons they adopted this principle of assuming the linear no-threshold model was more because of their concern about genetic effects rather than somatic effects. As to the other question, I'll try to answer it briefly. It's a complex issue. I touched on the transient worker issue--people moonlighting and moving from job to job in my remarks. But there's another element of this short-term contract workers. I do not like the term, and I don't like to use it, the idea of "burning out people." That is something that if it involves using inexperienced or unqualified workers is not really tolerable just on safety grounds, because the operations that people do on these plants that involve exposures typically are involved in the maintenance or repair of essential safety equipment. There is really no trivial exposure. So you want highly qualified workers to do these jobs. If a licensee began to run through unqualified workers, just piling up bodies to do jobs, it would be counter to safety in the first place, and secondly, would involve unduly large collective doses. If the regulatory strategy that's adopted emphasizes reducing collective doses, you would tend to catch this practice in that context, that is, the total number of men involved in doing a job is focused on and controlled, as well as exposures of individuals; you tend to root out this sort of practice which has occurred from time to time in the past of "burning out" individual workers.

Question: How did the NRC take that? Was that something they slapped the company on the wrist and said you shall not do this anymore?

Mr. R. Minogue: No, it's not contrary to the present regulations. I don't mean to give that impression. Let me give you a specific example. I think President Carter took part in an operation like this many years ago. It's conceivable that there might be some operation involved that required little specialized skill where a worker might be called on to go in and do one job, and then move out again and go back to his normal job outside the radiation field. It would be completely acceptable if that was part of a program that led to the minimum number of total man-rem, which, in my mind, I equate to total cancers. It's not that simple of a question to say, "Well, you should never do this." That, I think, is not a sensible answer. But rather to select whatever mode causes the least public impact. That's the basic bottom line. What is the best way to do a job that involves the least public health impact and accomplishes the safety goal? That's the test we use.

Dr. N. Wald: Excuse me, I think we'll have to let some other people ask questions.

John Blair: I'd just like to say one brief thing. It seems what I have heard today, as far as health effects of nuclear energy, is that's okay to shoot now and ask questions later because so much of this stuff we really don't know, and it seems that even this distinguished panel which is made up of academic and industry and government people doesn't have very many of the answers that the public is crying out for. With this kind of policy of making plenty of nuclear energy today, and not knowing what's going to happen with it in 10 years, is a policy that may get us into a lot of trouble in the long run.

I'd like to make a brief comment, if I may. I've been very impressed with what I've heard today in an area that is very new to me. However, I have to admit that the statistical projections that you gentlemen are forced to make, are forced to rely on, leaves me a little bit cold. It's not something I feel I can snuggle up with and really get to know. I am concerned about an area that I think is raised specifically by the nuclear power question and that's the area of health economics. I'm going to just scratch the surface in my comments, because if I do anything more, I'll reveal my profound ignorance of the subject matter. I am concerned with the extreme costs that appeared to be associated with nuclear power generation, particularly capital outlay, operating and maintenance, and also some of the internalized costs; hopefully, the costs to be internalized, such as waste disposal and the potentially staggering costs of decommissioning plants 30 or 40 years from now. There are two effects of that enormous cost. One is potential raising of the cost of electricity. I would refer anyone interested in that to the remarks made by the gentleman yesterday from the Edison Power Research Institute Electric Power, EPRI. Although I don't necessarily agree with the conclusions he drew, he did raise some questions about what are the societal and health impacts of increased electrical power costs. But mostly I'm interested in the fact that making the determinations as to what are the health impacts of nuclear power generation, can't be made in a vacuum that this discussion has been contained in. I'm not saying that that is a fault of the people who put together the program. It's inevitable what you're trying to do, but I think it is important to point out that we, as a society, have very limited resources and that if we decide to spend hundreds of millions of dollars or billions and billions and billions of dollars on nuclear power generation, that is money we're not going to be able to spend on health issues-- health promotion, other areas of health prevention, some of the fairly staggering cost of environmental control. I guess what I just want to point out is that the issues of human health costs of nuclear power generation are not just related to occupational and community safety, but also raise the issue of resource allocation as a determinant of health. I think that's a consideration of everything we've talked about in the last three days. But I think that, given the staggering costs that nuclear power may result in, that it is most sharply raised in this issue. I just wanted to point that out. Thank you.

Dr. N. Wald: Does anyone want to comment?

Panelist: I'd like to make several comments. I'm not sure that was a question. I think it was a short speech. First, I'll challenge the assumption that the cost of decommissioning a nuclear power plant is staggering in terms of the cost of the plant. That's simply not true. Much of the technology of decommissioning these plants is already established and, in fact, several have been decommissioned. But more important, the basic issue here that I am going to talk as a public health regulator. I found this conference an extraordinarily interesting experience. I had no idea how severe the health problems were in the coal industry until I came to this meeting. I would agree with and support any argument that said the public health impact of producing electrical energy and the use of electrical energy should be fully considered before one goes forward. But that means fully considered in all modes, and I've heard nothing to persuade me that the issues I try to grapple with in my day-in-day-out job, in terms of controlling radiation exposure both to the workers and the public, are being handled any less well than those associated with coal. I have had exactly the opposite impression. I've sat here and listened to people talk about sulfates and a complete uncertainty as to what the effects are, and some suggestions that "so let's not set any standards. We'll just keep on doing it." I've been a regulator for a long time, and I found that sort of discussion quite disturbing, personally.

John Blair: May I ask a follow-up question?

Dr. N. Wald: Let's give the panel a chance to respond. I really can't let anyone monopolize the question because there are other questions.

Panelist: I'd like to also respond to the decommissioning issue. There have been a number of studies by industry and by others about the cost of decommissioning and I have never seen anything that I consider to be a solid technical study that doesn't put the decommissioning cost of a nuclear power plant at a very, very low level when compared to the total capital outlay that you're talking about in a plant. As far as the economics of nuclear power, I think this is certainly variable, depending on where you are in the country, what the cost of coal, what the cost of hydro, the availability, etc. I don't see a lot of nuclear power plants being built where they're not economical. In fact, we don't see very many nuclear power plants being built, period, right now. And I believe if we don't get some built, I think we're going to have serious shortages in this country. And I say this also as a public health person and not just as a representative of the industry. I think that the public health benefits of adequate energy is an essential ingredient to the United States, and to our welfare and health overall. I guess that's about it.

Dr. E. Radford: I'd just like to add a very brief comment--not to prolong this discussion. It is absolutely true that the economic aspects of all these alternatives are important. This symposium was not designed to address these except in a very limited way, deliberately because we wanted the health implications given the prominence we think it has. I would agree that ultimately all of our activities that impact on either the general public or the workers, ought to be internalized in the cost of that activity. And I agree that it's far from being internalized. I don't think it's adequately internalized in the nuclear industry, for one case. For example, look at the mining problem which is still with us, and which is not being adequately factored into the equation, any more than the coal mining issue is in the coal cycle. So I think we've got to do it and one of the ways we do it, is by getting a better handle on the health effects, and that's what we're trying to do here.

Richard Ulrich, Group Against Smog and Pollution, Pittsburgh: I have a question concerning the exposure information for the people employed in the plants. Out of 70,000 workers, I think, the slide shows 11,000 workers receiving more than one rem. Now, I think, there are some people who do believe that one rem could be a sufficient standard, that is the standard should be lowered from 5 rems to 1 rem per year. Now, with those 70,000 workers, there were 30,000 or 25,000 who didn't get any exposure and certain others who are probably not working in the critical areas. Are these 11,000 out of a much smaller group of critical workers--are there steam fitters or some other group of whom there are only 10,000, and right now 8,000 or 9,000 of them actually are getting the 1 rem exposure or more. The implication of this health thing, if the standard is lowered, can nuclear power plants keep running or can most of them keep running, all of them keep running, or will they run out of workers?

Dr. N. Wald: Whom are you addressing? Whose slide was it you described? I think it was in the regulation.

Panelist: I think I can answer the question rather briefly. I think it's correct that of the group exposed to greater than the 1 rem cutoff, a large percentage of those are highly skilled workers that are associated with some of the critical welding and inspections operations. These skills are rather widely available throughout the country. It's a high level of skill that is relatively available, I think.

Richard Ulrich: So there will be workers available in other industries that the nuclear industry would be able to hire?

Panelist: The trouble is that I looked at this issue more from the point of view of what it is that produces the least public health impact. Running through more workers, if you could somehow make them available, might increase the total collective exposure. The perspective you're asking me to answer is more that of industry. I would assume they could train welders if all else fails. That wouldn't accomplish any public health goal.

R. Ulrich: I kind of suspect it's going to be hard to keep an industry going if it requires a heavy turnover of highly skilled individuals.

Panelist: I really doubt that very much. The cost of these repairs in terms of the worker cost is miniscule compared to the investment in these plants.

Ulrich: So they could keep a larger permanent work force? Maybe that would be a solution--it would have extra workers who . . .

Panelist: Workers of this type typically work for various contractors. They're not necessarily the employees of the utility.

Dr. N. Wald: We have time for one more question.

Jerry Rosen, University of Pittsburgh: My question to the panel is about the differences in estimated health effects from natural background which I raised earlier. I got the answer I expected from the panel. What concerns me is that a new interagency report which is in draft form, makes use of information on biological effects which is several years behind the times. It doesn't draw on the UNSCEAR Report of 1977. It obviously cannot draw on the new BEIR Report which, I belive, is due shortly. This is a document which I believe will be used strongly in the political decision making process related to future changes in radiation exposure limits. People like Mr. Minogue do not have some of this information available to them unless they go outside of their own agencies to gather it. I feel that this is detrimental to future policy making.

Dr. N. Wald: Anyone care to comment?

Dr. E. Radford: I'll just comment to your first point. I'm not quite sure what your last point was--that because we know more about radiation we should be less concerned about it, more concerned about it, or what? To answer your first question, I do agree that the decision on the part of the technical staff who prepared the Libassi Report to use BEIR I risks estimates was, in my opinion, a mistake. I'm not quite sure why they did it, except perhaps it was available to them in a form that they could use in their computer runs more readily than perhaps some of the other data that was possibly available to them. I think you're right. It is out of date and it is unfortunate that the BEIR report did not come out before the Libassi Report. In fact, I'm told that one of the reasons that the Libassi Report was delayed was because they were waiting for the BEIR Report to come out. I'm chairman of the BEIR Committee, and therefore I carry the onus of the fact that it isn't out. But we're doing our utmost.

New Speaker: The second point wasn't to say that we should go ahead with nuclear power or coal power, I think most of the decisions ahead of us are not really scientific. They are going to be political. I see issues being raised here by some of the questioners that are purely political and I don't think that the questions are very healthy in nature. Let's put it that way--at least the reasons for them being formulated.

Dr. N. Wald, moderator: I think we've run out of our time for questions and answers and I want to thank the speakers for their very lucid presentations and the audience for its contributions to the discussion. Thank you.

SESSION VI: FUTURE AREAS OF CONCERN

Wednesday afternoon, March 21, 1979

Moderator: Wesley W. Posvar, Ph.D.
Chancellor
University of Pittsburgh

POTENTIAL HEALTH PROBLEMS IN THE PRODUCTION OF SYNTHETIC FUELS FROM COAL

By
Maurice A. Shapiro, Professor

LONG TERM HEALTH IMPLICATIONS OF RADIOACTIVE WASTE DISPOSAL

By
William D. Rowe, Ph.D.

AREAS OF UNCERTAINTY IN ESTIMATES OF HEALTH RISKS

By
Leonard D. Hamilton, M.D., Ph.D.

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POTENTIAL HEALTH PROBLEMS IN THE PRODUCTION OF SYNTHETIC FUELS FROM COAL

By

Maurice A. Shapiro, Professor
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Introduction

The first commercial coal hydrogenation (liquefaction) plant was built at Leuna, Germany in 1926. With the evolution in design improvements for this plant, liquid fuel products were generated which were comparable in composition to those derived from petroleum. Twelve additional German coal hydrogenation plants were eventually built so as to compensate for the lack of that country's oil and gas supplies. It was these hydrogenation plants that during World War II supplied over 85% of the aviation gasoline used by the Luftwaffe. On March 8, 1979, the N.Y. Times reported that the Sasol plant under construction in Secunda, South Africa will, in 1982, process 27 million tons of coal a year and produce 500 million gallons of liquid fuel per year. The plant will employ 7,500 workers.

One of the key goals of the Administration's energy planning has been the achievement of greater American independence from foreign oil and gas supplies. The United States currently utilizes oil for 46% of its energy needs, natural gas for 32%, while coal provides only 17% (1). Since our known U.S. coal reserves (8000×10^{15} Btu) far outweigh the U.S. oil reserves (200×10^{15} Btu), it is evident that the United States will have to use coal or coal conversion products as a major means of reaching such energy independence (2).

Several governmental agencies have reported that the U. S. coal reserve base is approximately 400 billion tons. The Department of Energy has estimated that by the year 2000, high- Btu gasification will supply 6.8×10^{15} Btu's and low-Btu gasification 1.8×10^{15} Btu's of energy (3).

Approximately 176 sites have already been identified in the U.S. each of which have sufficient coal and water (large quantities of water are required in the conversion process) to support the operations of a gasification plant (250 Mscf/day) for twenty years. If coal gasification and liquefaction processes are expected to supply such vast quantities of energy in the near future, it is imperative that potential health impacts associated with synthetic fuel production be determined.

The aim of this paper is to evaluate the potential impacts of coal conversion derived pollutants as they are transported and transformed in air, water and soil and to ascertain the public and occupational health consequences.

DRINKING WATER - POSSIBLE HEALTH EFFECTS

The effluents from coal conversion processes are numerous, with the majority of streams being relatively clean and thus amenable to recycling and/or safe disposal. Some of the residues are contaminated by toxic and mutagenic substances and must be treated before discharge. The generation of these by-products depends upon the interactions of major factors in the design and operation of the coal conversion plant, such as operating temperature, method of coal injection, and coal type. These factors will affect the composition and ultimately the potential health impacts of effluent streams derived from gasification and liquefaction facilities. Based upon information obtained from existing pilot plants, a full scale commercial gasification plant (250 Mscf/day of pipeline gas) may be expected to generate between 0.4 and 1.2 million gallons of liquid discharge per day. As an example of the range of contamination loadings, a coal gasification plant generates approximately 50-100 lbs of tar, 30-70 lbs of oil and naptha, and 8-12 lbs of phenols per ton of coal (4).

Some other constituents expected to be present in the wastewater include ammonia, cyanates, thiocyanates, arylamines, aliphatic hydrocarbons, mono- and polycyclic hydrocarbons, organic-sulfur compounds, carboxylic acids, esters, furans, tetralins and organometallic complexes.

To assess possible pollutant concentrations which may enter potable water supplies and thereby affect health, it is necessary to investigate pollutant removal efficiencies of various treatment methodologies. Methods which could be utilized to treat coal gasification wastewater include: ammonia stripping, solvent extraction of phenols, biological oxidation of phenols and other organic constituents, and possibly carbon adsorption and chlorination. Ozonation will probably not play a major role in the wastewater treatment scheme due to its high cost and low efficiency (5).

The concentration of pollutants remaining after wastewater treatment are shown in Table 1. This information is based upon data obtained from operating pilot coal gasification wastewater treatment plants or by extrapolation of results derived from treatment plants serving similar industries. Using the concentrations in Table 1, it is possible to calculate constituent concentrations of pollutants when dispersed and diluted in specified receiving streams (Table 2). We chose specific locations on the Allegheny and Monongahela Rivers to reflect possible future sites for coal gasification facilities. As can be seen in Table 2, during average flow conditions, the constituents will probably be diluted to such an extent that their contribution to environmental concentrations will be negligible. However, during 7-day, 10-year low flow conditions, some constituents will not be sufficiently diluted so as to comply with various stream quality standards. For instance, during the 7-day, 10-year low flow of the Monongahela River at Charleroi, Pa., the increase of phenol concentration above background in the receiving water exceeds the recommended

1ppb standard for surface waters (6). Table 2 does not take into account the background levels of constituents in receiving streams. It is possible that the increase of some constituents due to coal conversion wastewater when added to background concentrations may cause them to exceed water quality standards.

Even though the load of pollutants, when diluted with receiving stream water will generally be reduced to very low levels, review of the literature indicates that there are some classes of compounds which warrant concern. The classes of major concern are organic contaminants, cyanate and thiocyanate, ammonia and hydrogen sulfide.

Organic Contaminants. Monohydric and dihydric phenols are major organic compounds in wastewater generated during coal gasification. These phenols can be reduced to trace concentrations by biological oxidation. Phenols in drinking water lead to significant taste and odor problems and possible health effects. It should be noted that chlorination of phenol containing water usually increases taste and odor problems due to the lower odor thresholds associated with chlorinated analogs of certain phenols. Polycylic phenols are present in lower concentrations than monocyclic phenols. The efficiency of removal of polycyclic phenol by biological oxidation is less than that obtained for mono-and dihydric phenols.

Monocyclic and polycyclic N-aromatics are present in small quantities in coal gasification wastewater. The ingestion of pyridine (a major compound in this class of organics) has been shown to cause liver and kidney damage (9). Aliphatic acids may be present in coal conversion wastewaters at concentrations up to 700 mg/l, with acetic acid comprising approximately 85% of this aliphatic acid section. In sufficiently dilute form, acetic acid is not toxic nor are its acetates, such as potassium and sodium acetate.

Cyanide and Thiocyanate. Data on cyanide and thiocyanate concentrations in processed coal gasification wastewater is needed because of these compounds' toxic properties. There are inconsistencies in the literature regarding cyanide and thiocyanate quantities generated by gasification operations. The same is true of the cyanide removal efficiencies of several treatment methods. Cyanide is present in coal gasification wastewater at concentrations ranging from 0.01 and 2.28 mg/l, and thiocyanate is present in raw coal gasification effluent in concentrations ranging from 20 to 400 mg/l. Given an average concentration of thiocyanate (150 ppm) and a practical removal efficiency of 70%, then approximately 45 ppm of thiocyanate is expected to be present in treated coal gasification wastewater. The average concentration of cyanide in such treated wastewater is 0.6 mg/l. Reduction of cyanide concentrations in wastewater has been reported to be as high as 90%. In general practice, reduction of cyanide by biological oxidation is approximately 60%. Cyanide at low concentrations (10 mg/l or less) is converted in the human body to the less toxic thiocyanate species (8). Upon chlorination, either at a coal gasification plant or at a municipal water treatment plant, cyanide is easily oxidized. The remaining concentration is below the health effect threshold level.

Ammonia. Approximately 80% of the nitrogen in the feed coal is converted to ammonia during coal gasification. Some 20-22 lb of ammonia are generated per ton of coal gasified. The average concentration of ammonia in gasification wastewater is 7500 mg/l. Ammonia stripping, lime treatment and biological oxidation are expected to reduce ammonia concentrations in treated coal gasification wastewater to a range of 5-15 mg/l.

The presence of ammonia, nitrite and nitrate in potable water supplies results in a variety of pollutional problems. Ammonia and its oxidized analogs are potential algal and microbial nutrients in water distribution systems. Ammonia also increases the chlorine demand at water treatment plants which practice free-residual chlorination. In addition, it increases the biochemical oxygen demand in receiving waters.

The conversion of ammonia to nitrite and nitrate is a significant occurrence due to the potential adverse effects of these compounds on human health. The well known health hazards of nitrites and nitrates in drinking water are the induction of methemoglobinemia (especially in infants) and the potential formation of carcinogenic nitrosamines. In infants, nitrite acts in blood to oxidize hemoglobin to methemoglobin, a form which cannot act as a carrier of oxygen to the tissues leading to anoxia and possible death. The reported concentration of nitrate in water needed to induce methemoglobinemia varies from reference to reference. However, very few cases have been related to drinking water containing less than 10 mg/l nitrate as nitrogen (11). It is known that infants have been fed water that contained larger amounts of nitrate without developing methemoglobinemia.

To assess the impact of nitrate in drinking water as a causal agent of methemoglobinemia, one must determine what other sources of nitrate in the environment will be ingested. An "average" person has a total daily nitrite-nitrate intake of approximately 110 mg, with vegetables contributing over 80% of the total input (12). Water, on the other hand, contributes no nitrite and 0.7% nitrate as compared to other sources.

Upon dilution with stream or process water, the concentration of ammonia would be considerably less than the 5-15 mg/l present in the treated coal gasification wastewater. Assuming that all of this ammonia is converted to nitrate, it is likely that the resulting concentration would be below the threshold for methemoglobinemia. However, when the contributions of nitrate and nitrite from diet, air and water supplies are added, the amount of ammonia present in coal gasification effluents which is then converted to nitrate and nitrite may exascerbate existing problems.

Adverse health impacts may also ensue when nitrates are converted to carcinogenic N-nitroso compounds in water. The contribution of nitrate transformed to N-nitroso compounds in water compared with the amount contributed by food ingestion has not been sufficiently well determined. However, epidemiological studies have correlated the increased incidence of gastric cancer with elevated nitrate levels (90-110 mg/l)in drinking water (13).

Hydrogen Sulfide. Hydrogen sulfide (H_2S) has been reported as being present in untreated coal gasification wastewater in a range of concentrations from < 5 mg/l to 250 mg/l. Biological oxidation and acid gas stripping have been reported to be 97% and 99% effective in removing H_2S from wastewater.

The toxic properties of H_2S are exerted in the undissociated form. The extent of dissociation is largely dependent upon pH, with approximately 50% dissociation occurring at pH 7.0. If oxygen is present in the water at concentrations above 0.025 mg/l, then H_2S will probably be converted into sulfate. If sulfide is not converted into sulfate, it would most likely form insoluble metal sulfide complexes due to its strong ligand properties.

SOLID WASTES - POSSIBLE HEALTH EFFECTS

Solid wastes along with contaminated liquids are generated during coal conversion operations. Solid residues from coal gasification include ash and char from the gasifier, desulfurization and wastewater treatment sludges, and disposable catalysts. The volume of solids generated depends on the characteristics of the feed coal and the conversion process. The literature reports that typically a 250 Mscf/day gasifier will produce 3000-4000 tons/day of ash and char (14). These gasification solid wastes contain higher concentrations of trace elements than the feed coal. Somerville et al. (15) have shown that 81% by weight of the major and trace inorganic elements in the feed coal exit with the ash. In the coal ash, the major constituents are complexes of aluminum, calcium, iron, potassium, magnesium, sodium and silicon.

The potential for trace element contamination of ground or surface water by leachates derived from land-filled or ponded ash depends on the chemical and physical characteristics of the stored sludge and ash, its rate of application, the soil characteristics and the hydrological regime of the area. Uptake and toxicity to flora and fauna (especially microorganisms) must be considered. The transport of trace elements in the soil depends upon the rate of water movement, adsorption capacities and chemical reactions taking place within the soil profile.

Trace elements sorbed on soil will be strongly held by some soils and thus prevent rapid leaching of many elements into surface and ground waters. Factors affecting trace element attenuation by soil include pH, oxygen availability, soil particle size distribution, cation exchange capacity, amount and type of hydroxides and oxides of Fe, Mn, and Al, pore size, and concentration of organic material, salt, and microorganisms.

The literature emphasizes a concern about the presence and behavior in coal gasification ash of the following elements: arsenic, cadmium, chromium, fluorine, mercury, sulfur, and selenium.

Arsenic. Health effects have been associated with the ingestion of drinking water with high concentration of arsenic. In Antofagasta, Chile, a city with a population of 100,000, drinking water was reported to contain a weighted average arsenic concentration of approximately 600 g/l (16). This high arsenic concentration was linked to a high incidence of cutaneous skin lesions (145.5/100,000 for males and 168/100,000 for females). After the installation of a water treatment plant, the arsenic content of the water was reduced to 80 ug/l, while the incidence of cutaneous skin lesions dropped to 9.1/100,000 for males and 10/100,000 for females. Due to the low concentration of arsenic present in coal gasification ash, it is expected that most soils will have the ability to attenuate arsenic sufficiently through such mechanisms as coprecipitation with sulfide, ferric hydroxide, ferric chloride, and ferric sulfate.

Cadmium. Food is the primary source of cadmium intake for man. The total daily intake of cadmium from air, water, food, and tobacco ranges from 40-190 ug/day. Drinking water contributes only a small fraction (less than 5%) of the total (11). A variety of disease states are thought to be influenced or caused by high levels of cadmium in various organs. Chronic ingestion of cadmium at levels of greater than 600 ug/day has been reported to be responsible for the onset of "Itai-Itai" disease in Japan (11). Along with its kidney toxicity, there has also been some indications in animal studies that cadmium is carcinogenic and/or teratogenic. Cadmium is present in gasification ash at a concentration of approximately 0.2ppm. Cadmium also has a very low solubility in water. Due to these two factors, it does not seem likely that cadmium in coal gasification and liquefaction ash would contaminate drinking water supplies at levels which cause disease.

Chromium. Concentrations of chromium in natural waters are limited by the low solubility of chromium (III) oxides. Very little information is available on the average total daily intake of chromium, although it appears to be slightly lower than the intake for cadmium. On the average, approximately 0.04% (400 ppm) of coal gasification and liquefaction ash is composed of chromium. This is a significant fraction given the huge amount of ash expected to be generated. There are many inconsistencies in the literature regarding the renovation potential of soil for chromium. However, even though chromium is relatevely insoluble in water, it is considered a potential hazardous compound in ash due to the many uncertainties about its behavior in the environment.

Fluoride. Fluoride, due to its anionic nature, is not attenuated to any great degree by the soil. The lethal dose of sodium fluoride for man is 5 g (17). We know that teeth and bone are the most fluoride sensitive tissues. National Academy of Sciences (11) have concluded that symptomatic skeletal fluorosis can occur at a level as low as 3 ppm. They have also concluded that the possibility of fluoride causing adverse effects such as allergic responses, mongolism, and cancer has not been adequately documented.

Sulfur. Sulfur is one of the major components of coal gasification ash. In anaerobic environments, sulfur is usually present as sulfide ($S^=$). In this species, it can easily form insoluble precipitates. However, under aerobic conditions, sulfur is oxidized to sulfate ($SO_4^=$). National Academy of Sciences (11) have concluded that no adverse health effects have been noted when the concentration of sulfate in water is less than 500 mg/l. The observed physiological effect at concentrations greater than 1,000 mg/l is the induction of diarrhea.

Selenium. Selenium is easily volatilized during gasification and emitted to the atmosphere. Therefore, the amount of selenium in coal gasification ash is very small and other sources of selenium (e.g. diet) will overshadow the possible contribution of selenium from coal gasification solid residue. Mercury, likewise, is easily volatilized and can be emitted as a vapor from coal gasification stacks. Therefore, its environmental effect due to its presence in ash or sludge will be minimal.

HEALTH IMPLICATIONS OF AIR POLLUTANTS

Potential atmospheric pollutants from synthetic fuel production arises from the auxillary operations as well as the actual conversion process. A list of some of the potentially hazardous substances follows in the section dealing with occupational health problems. These substances may be present in coal conversion process streams; however, because of the enclosed and pressurized nature of a gasification or liquifaction system, air emissions during regular plant operations are expected to arise mainly from the auxillary operations. During startup or in an emergency, emissions could be of high rates but of short duration and, for high-BTU gasification, are expected to be less than one percent of the total plant air emissions. Emissions from the gasification process itself would arise mainly from leaks in pump seals, joints, flanges, compressors, etc., and from venting operations (18). Quantities of the major gaseous effluent streams from the various processes are compared in Table 3 with the corresponding streams from a large power plant using recycled cooling water (19).

Because published data on emissions from coal conversion plants is sparse (20) only auxillary operations were considered in the ERDA Synthetic Fuels Commercialization Program Report (21) which estimated air emissions from coal conversion plants. In order to make the resulting pollutant loads meaningful, they were calculated for a conversion facility that would produce enough fuel to support a 1000 MW power plant operating at 33 percent efficienty. Table 4 indicates ambient air quality contributions of such a plant. National Ambient Air Quality Standards are shown in the headings (21). Of interest is the relative contribution of gasification plants to ambient air quality. The ratio of sulfur dioxide derived from the gasificiation plant to the Annual Average Standard ranged from 0.01 to 0.24; the NO_2 ratio ranged from 0.01 to 0.22; particulates from 0.0 to 0.3; and hydrocarbons ranged from 0 to 0.04 of the Three Hour Maximum Standard. Some of these amounts are important contributions to the ambient load. The median and 90th percentile values for total suspended particulates gathered by State and local air pollution control agencies are presented in Table 5 along with the secondary National Ambient Air Quality Standard Values (22). Since the ambient air quality in certain situations already exceeds the standard, the contribution of such a gasification plant could add to the already high ambient levels.

Experimental "Hygas" bench scale results indicate that much of the total flow of trace elements would get into downstream process systems and eventually be removed in the gas quench, shift conversion, acid-gas removal, or on catalyst surfaces. However, data from the Synthane pilot plant gasifier indicate that a major fraction of trace elements concentrate in the char residue which may be burned in the utility boiler, releasing them to the atmosphere upon combustion. Therefore, the amount of trace element concentration would be increased 4 to 5 times over that normally emitted by a coal fed boiler of similar-size (23). The background concentration of several metals and the contribution from possible coal use projected in the year 2020 is given in Table 6; potential violations of suggested acceptable atmospheric trace element concentrations are primarily due to already higher background levels. In a report of the Argonne National Laboratory on coal utilization in the Illinois River Basin, it is stated that the gastrointestinal tract, rather than the respiratory system, could be the most significant route of entry for coal related air and waterborne trace elements. The significance of the gastrointestinal tract is further enhanced if dietary trace element intake were added. Moreover, uptake of trace elements in food materials due to higher ambient levels in air may increase such a contribution. In spite of this, projected coal utilization is not anticipated as a prime contributor of trace element body-burden, but it could account for as much as one-third of the vanadium and one-tenth of the chromium body burden in exposed populations (24).

Combustion of fossil fuels accounts for a substantial portion of atmospheric contamination by mercury. Studies of persons occupationally exposed indicated a dose-response relationship involving the nervous system starting with concentrations as low as 0.1 mg/m^3. Mercury, therefore, in ambient air probably represents little if any health problems for the general population (25).

Also to be considered is the radiological impact of emissions due to conversion processes. Based on results of analyses of coal, SRC solid fuel, and flyash samples from both, the following observations were made:

1. Uranium levels were less than thorium in all cases except for SRC particulates

2. Uranium and thorium were more concentrated in particulate samples than in the coal and SRC solid fuel

3. Literature cited in the report (26) indicated that at least 90 percent of the uranium is expected to be retained in the bottom ash and collected fly ash

The estimated level of U-238 expected to be discharged from a power plant which combusts SRC solid fuel is 0.2 ug/m3 which is an order of magnitude less than the 7 ug/m3 "general public" exposure standard for air (26).

Although many potentially hazardous materials are residues of the coal conversion process, there is no direct, continuous atmospheric emission of these substances (27). Concentrations of some organic compounds found in an urban atmosphere, and also suspected present in coal conversion process streams are shown in Table 7. Polycyclic aromatic hydrocarbons (PAH) can be readily absorbed by animals through ingestion, inhalation, or skin contact (29); the carcinogenicity of PAH adsorbed onto polycyclic organic material (POM) through inhalation has been documented with experimental animals (30). However, in 1970,BaP concentrations in over 100 United States cities were one to two orders of magnitude less than 0.12 ug/m3, the suggested maximum concentration of atmospheric BP (31). Since the coal conversion process is expected to be a more controlled system than coking, its pollution contribution should be less.

OCCUPATIONAL HEALTH CONSIDERATIONS

The first occupational cancers were reported as far back as 1775, when the prevalence of scrotal cancer among British chimney sweeps was determined to be eight times that of the general populace (32). Chimney soot was identified as the suspected carcinogen. One hundred and thirty-two years later, England officially acknowledged epitheliomatous cancer or ulceration from exposure to pitch, tar, and tarry compounds as valid grounds for workmen's compensation (33, 34). Today, industrial materials such as soot, shale oil, certain aromatic amines, coal tar, and isopropyl oil, among others, are recognized occupational carcinogens (35, 36). Epidemiological evidence has conclusively shown that workers routinely exposed to the combustion and/or distillation products of bituminous coal run an increased risk of developing cancer of the skin or viscera (37, 38, 39).

Although large-scale coal hydrogenation plants were operated in Germany in the 1920's, Britain in the 1930's and South Africa in the 1950's, the best insight we have into the potential health effects of coal conversion is the result of a pilot plant operated at Institute, West Virginia during most of the 1950's. This Union Carbide facility produced chemicals via coal hydrogenation (liquefaction) with a design capacity of 300 tons coal/day. Even before the Institute Plant "went on line" in 1952, it was known that the facility would generate a wide variety of chemicals, many which would pose potential acute or chronic health hazards to the workers. Over two hundred individual chemicals were eventually identified from the Institute plant (40), the majority of which fit into the following six classes of compounds:

a.) aliphatic hydrocarbons
b.) single ring aromatics
c.) polynuclear aromatics
d.) phenols
e.) aromatic amines
f.) N-heterocyclic aromatics

However, several inorganic gases as well as dusts and particulates were also found to be present in the plant environment.

During the first two years of operation several intermediate and final products of the Institute facility were painted on the skin of mice to test for tumorigenicity (41). The light oil stream and its eight fractions did not exhibit any tumorigenic activity. As a rule, these light oil stream fractions have relatively low boiling ranges, phenolic pitch being the only component separating above 260°C. Conversely, the process streams and residue boiling at higher temperatures (middle oil stream, light oil stream residue, pasting oil stream, and pitch product stream) were all demonstrated to be highly carcinogenic to the skin of mice. Carcinogens implicated by these results include polycyclic aromatics such as phenanthrene (bp 339°C), perylene(sub 350-400°C), picene (bp 520°C), and pyrene (bp 404°C) (42). The structural formulae of these and other high boiling, polycyclic aromatic constituents of coal-tar are depicted in Figure 1.

The Institute work force was thoroughly warned of the results of the animal studies in late 1952 and 1953. Moreover, the workers were provided with safety education and changes of clothing during work. Despite these precautions, cases of skin cancer were discovered among the work force. Dr. R.J. Sexton, the plant's medical director, examined for five years the 359 men regularly assigned to work the plant area. Sexton found nine workers with one cutaneous cancer each and one worker with two cutaneous cancers as shown in Table 8. He also found forty workers with one cutaneous precancerous lesion each as shown in Table 9 (34, 44). This incidence of skin cancer is many times that of either West Virginia or the United States (34). These skin abnormalities were believed to have resulted from occupational exposure to coal tar, pitch, or high boiling-polycyclic aromatics primarily via air-borne (vapor or condensed droplet) contamination routes (45, 46).

In a study conducted by the Stanford Research Institute (44) nine of the ten workers described in Table 8 and all forty workers listed in Table 9 were followed-up in order to determine their vital status as of July 1977. Of the ten workers originally diagnosed with skin cancer, two died in the intervening period from causes other than cancer, two retired (one of whom is ill with lung cancer), five were still working, and one subject could not be found. Of the forty workers diagnosed with pre-cancerous skin lesions, three died from causes other than cancer, thirteen retired, twenty-three were still working, and one retiree was hospitalized with Parkinson's disease and prostate cancer. The Stanford researchers note:

> ..... that the available data do not support the initial hypothesis; that those workers exposed to heavy streams of toxic materials from the coal hydrogenation process, with evidence of cancerous skin lesions, may be at increased risk of developing systemic carcinoma. This observation is based upon a marked lack of cancer-related deaths or morbidity in both the confirmed skin cancer group and the precursor group, after a latency period of 18-20 years.

Nonetheless, two additional facts merit consideration - a.) that the cohort under examination was small; and b.) that the 309 workers who did not exhibit lesions in the Sexton study (34) were not examined in the Stanford study (44). Few health effect studies, other than the one conducted at the Institute facility, have been reported. Hueper (47,48) tested the carcinogenicity of Bergius and Fischer-Tropsch oils obtained from the experimental coal hydrogenation-liquefaction facility at Bruceton, Pennsylvania. Fractions were tested by repeated application to the skin of mice and rabbits, and I.M. injections to the thighs of rats. Eight of nine Bergius oil fractionation products were found to be carcinogenic, the degree of carcinogenicity usually increasing with boiling points. Fischer-Tropsch products were less carcinogenic and seemed to have a narrower species and tissue susceptibility spectrum. Epidemiological studies of gasworks and coking operations (38, 39, 49, 50) have provided some insight into the potential health effects of coal conversion plants because the types of toxic chemicals present are similar. However, coal conversion plants operate by necessity with good containment and worker exposure to these agents is projected to be much less than in the aforementioned industries (42).

Researchers at Battelle-Columbus Laboratories (43) examined the potential occupational health effects of three gasification processes: the high-pressure, fixed-bed system; the atmospheric-pressure, entrained-suspension system; and the atmospheric-pressure, fluidized bed system. In all of the gasification schemes the greatest carcinogenic potential was determined to exist in the early process stages, when the complex organic constituents of coal undergo structural degradation. It is during these early operations that the risk of spills or leaks must be minimized. In the later stages, the output of the gasifier approaches a typical gas in composition and the carcinogenic potential is eliminated or minimized. Coal liquefaction processes pose a greater danger to the workers. Sophisticated engineering and industrial hygiene programs will be required in order to minimize worker contact, both dermal and respiratory, with organic compounds such as phenanthrenes, pyrenes, 1, 2-benzanthracenes, chrysenes, and five-ringed compounds.

We believe that worker exposure at future commercial-scale, conversion facilities will be maximal during shutdowns or equipment modifications/replacements attempted during periods of operation. Under such circumstances workers may be required to open process lines or enter vessels. It is worth noting that most of the skin cancer subjects reported by Sexton and shown in Table 8 were engaged in plant maintenance at the Institute, W. Va. facility. Our major concern is the exposure of such maintenance workers to the known carcinogens and cocarcinogens listed in Table 10. Most of the other chemicals present, as well as physical agents (heat and noise), can be dealt with by good industrial hygiene practice.

Table 1. Representative Water Pollutant Loadings from a 250 Mscf/day Synthetic Natural Gas Plant Using Illinois #6 Coal

Parameter	Untreated		Treated(a)		Efficiency of Treatment Based on Loading Reduction (%)
	Conc. (mg/l)	Loading (lb/day)	Conc. (mg/l)	Loading (lb/day)	
TSS	600	16,500	20	550	97
pH	8.6	-	8.5	-	-
Phenols	2,600	71,500	0.4	10	99+
Oil	7,500	13,800	5	138	99+
COD	15,000	413,000	90	2,500	99
BOD	2,300	62,400	14	375	99
NH_3	8,000	220,000	7.5	206	99+
Cyanide	0.6	16.5	0.1	2.75	83
Total Solids	1,400	38,500	12	330	99
Thiocyanate	150	4,130	45	1,239	70
Phosphate as P	2.5	69	0.3	8.3	88
Chloride	500	13,800	25	688	95
Fluoride	56	1,540	6	165	89
SO_4	-	39,000	12.1	334	99
Fe	3	82.5	-	-	-
Pb	3	82.5	-	-	-
Mg	2	55	-	-	-
Zn	0.06	1.65	-	-	-
As	0.03	0.83	-	-	-
Cu	0.02	0.55	-	-	-
Cr	0.006	0.16	-	-	-
Cd	0.006	0.16	-	-	-
Mn	0.04	1.1	-	-	-
Ni	0.03	0.83	-	-	-
Al	0.8	22	-	-	-
Se	0.36	9.9	-	-	-
Ba	0.13	3.6	-	-	-

(a) Flow of condensate is approximately 5.0 cfs

Source: Adapted in part from Argonne National Laboratory (7).

Table 2. Coal Gasification Wastewater Constituent Reduction Via Receiving Stream Dilution

Constituent	Treated Condensate Concentrations (mg/1)(a)(b)	Concentration (mg/1) of constituent after dilution with receiving streams of the following flow rates (c):			
		Monongahela River at Charleroi, PA		Allegheny River at Kittanning, PA	
		7-day, 10-year low flow (420 cfs)	Average flow (8975 cfs)	7-day, 10-year low flow (2,700 cfs)	Average flow (15,570 cfs)
TSS	20	0.23	0.0010	0.037	0.0063
Phenols	0.4	0.005	0.0002	0.0007	0.0001
Oil	5	0.06	0.003	0.01	0.002
COD	90	1.0	0.05	0.2	0.03
BOD	14	0.16	0.0077	0.026	0.0044
NH_3	7.5	0.087	0.0041	0.014	0.0024
Cyanide	0.1	0.001	0.00006	0.0002	0.00003
Total Solids	12	0.14	0.0066	0.022	0.0038
Thiocyanate	45	0.52	0.025	0.083	0.014
Phosphate as P	0.3	0.003	0.0002	0.0007	0.0001
Chloride	25	0.29	0.014	0.047	0.0081
Fluoride	6	0.07	0.003	0.01	0.002

(a) Assumed condensate flow of approximately 5.0 cfs

(b) Values obtained from Reference (7)

(c) Receiving stream water assumed to contain zero concentration of parameter before addition of condensate; complete dispersion is also assumed; 7-day, 10-year low flow and average flow values were obtained from Kay et al. (8).

TABLE 3. MAJOR GASEOUS EFFLUENT STREAMS (19)

Processes	Gaseous effluent streams, ton/day		
	CO_2 + Flue Gas[a]	C.T. Air[b]	Nitrogen[c]
Low- and medium-Btu gasification	4,900-19,200	600,000-2,221,000	0-38,000
High-Btu gasification	45,000-94,000	546,000-3,264,000	0-21,000
Liquefaction	39,000-66,000	1,250,000-2,655,000	6,000-16,000
1,000 MWe power plant (example)	110,000	3,500,000	0

SOURCE: Magee (19)

[a] Contains combustibles, sulfur compounds, and oxides of nitrogen.

[b] Effluent cooling tower air may contain volatile organic or inorganic compounds present from leaks or other sources.

[c] Waste nitrogen streams from oxygen plants are usually clean.

TABLE 4. AMBIENT AIR QUALITY CONTRIBUTIONS OF UNIT HIGH-BTU FIXED-BED GASIFICATION PLANT (21)

Region/Distance	Ambient Air Concentration (μg/m^3)						
	Sulfur Dioxide			Nitrogen Dioxide	Particulates		Hydro-carbons[(c)]
	Annual Avg. (STD 80) μg/m^3)[(a)]	24-Hr. Max. (STD 365) μg/m^3)[(a)]	3-Hr. Max. (STD 1300 μg/m^3)[(b)]	Annual Avg. (STD 100 μg/m^3)[(b)]	Annual Avg. (STD 60 μg/m^3)[(b)]	24-Hr. Max. (STD 150 μg/m^3)[(b)]	3-Hr. Max. (STD 160 μg/m^3)[(b)]
Appalachia							
1 km from plant							
Stability Class B	19	61	92	19	4	12	4
Stability Class D	1	3	4	1	18	60	0
5 km from plant							
Stability Class B	8	23	34	3	0	1	1
Stability Class D	11	34	50	10	3	9	2
Eastern Interior							
1 km from plant							
Stability Class B	19	65	99	22	5	16	6
Stability Class D	1	3	4	1	16	52	1
5 km from plant							
Stability Class B	8	27	38	4	1	2	1
Stability Class D	11	34	53	11	2	9	3

(a) These values are the primary National Ambient Air Quality Standard values.
(b) These values are the secondary National Ambient Air Quality Standard values.
(c) Hydrocarbons are a precursor to the formation of photochemical oxidants. The 1-hour secondary National Ambient Air Quality Standard for Oxidants is 160 μg/m^3,

Stability Classes refer to atmospheric stability; Class B is unstable; Class D is neutral. Highest pollutant emission rates which resulted from various coals were used in these calculations of ambient air concentration. Possibly used control efficiencies of 99,7% for particulate removal, 75% NOX removal, 85% FGD, 95% for sulfur recovery,

Adapted from ERDA (21).

TABLE 5. 1976 CONCENTRATIONS OF TOTAL SUSPENDED PARTICULATES (22)
(μg/m3)

	Median	90th
Annual Mean (2350 sites)	66	90
Peak Daily (2350 sites)	165	325
Region III Annual Mean[A]	55	90
Region V Annual Mean[B]	60	95
Annual Average (Sec. NAAQ STD)	60	
24-Hour Maximum (")	150	

[A] Pennsylvania, West Virginia, Virginia

[B] Ohio, Indiana, Illinois, etc.

Adapted from U.S.E.P.A. (22).

TABLE 6. BACKGROUND URBAN AND NON-URBAN AIR QUALITY AND PROJECTED 3-HOUR AND 24-HOUR AIR QUALITY DUE TO PROJECTED 2020 COAL UTILIZATION IN THE ILLINOIS RIVER BASIN ($\mu g/m^3$)

	As	Be	Cd	Cr	Cu	Fe	Hg	Mn	Ni	Pb	Se	V	Zn
Continuous Exposure Standard	0.17	0.0066	0.17	0.33	0.33	-	0.03	16.5	3.3	0.5	0.66	0.17	3.3
Background Concentrations[a]													
Annual average													
Urban Air	-	.00002	.0025	.0054	.106	1.17	-	.032[b]	.082	.886	-	.010	.32
Non-urban Air	-	.00001	.0001	.0028	.1264	.24	-	.007	.001	.578	-	.001	.10
Concentrations due to 2020 Coal Utilization													
24-hr. maximum	.00056	.000064	.00015	.00056	.00012	.21	.000013	.00098	.00054	.0029	.00032	.00087	.0058
3-hr. maximum	.029	.0033	.008	.029	.0063	10.9	.0067	.051	.028	.152	.017	.046	.304

[a] From G. Ackland et al., "Air Quality Data for Metals 1970-1974 from the National Air Surveillance Networks," Environmental Monitoring and Support Lab, Research Park, North Carolina, EPA-600/4-76-041, August 1976.

[b] Environmental Health Resource Center suggests a 24 Hour Average of 0.006 ug/m^3 based on synergram with sulfur dioxide.

Adapted from Argonne National Laboratory (24).

TABLE 7. CONCENTRATIONS OF SOME LARGE ORGANIC COMPOUNDS IN THE AVERAGE AMERICAN URBAN ATMOSPHERE (28)

Compound	Airborne Particulates µg/m
Benzo (a) pyrene	0.0057
Benzo (e) pyrene	0.005
Fluoranthene	0.004
Perylene	0.0007
Benzo (g, h, i) Perylene	0.008
Coronene	0.002

SOURCE: Sawicki (28)

TABLE 8 : CUTANEOUS CANCER CASES IN 359 COAL HYDROGENATION WORKERS* EXAMINED FOR FIVE YEARS

Case	Subjects Age	Job Assignment	Length of Exposure (months)	Body Site	Diagnoses	
					Local Pathologist	Out-of-Town Pathologist
1	30	Operations	41	Forearm, right	Basal-cell carcinoma	Malherbe's calcifying epithelioma
2	33	Maintenance	62	Cheek, left	Basal-cell carcinoma	Basal-cell epithelioma
3	29	Maintenance	12	Cheek, left	Squamous-cell carcinoma	No pathology done, clinical diagnosis only
4	37	Maintenance	48	Buttock, left	Squamous-cell carcinoma	Inverted follicular keratoma type of seborrheic keratosis
5	43	Maintenance	108	Hand, left	Squamous-cell	Squamous-cell carcinoma
5	43	Maintenance	132	Ear, right	Mixed basal and Squamous-cell carcinoma	Metatypical carcinoma
6	34	Operations	83	Hand, left	Intraepithelial squamous-cell carcinoma	Bowenoid keratosis
7	46	Operations	83	Neck, posterolateral, right	Squamous-cell carcinoma	Prickle-cell epithelioma
8	40	Operations	116	Ear, right	Squamous-cell carcinoma	Keratoacanthoma
9	33	Maintenance	60	Leg, left	Keratosis	Intraepithelial squamous-cell carcinoma
10	38	Maintenance	72	Leg, right	Keratosis	Squamous-cell carcinoma

* White Males Only

Source: Sexton (34)

TABLE 9

SUMMARY OF PRECANCEROUS CUTANEOUS LESIONS AMONG 359 COAL HYDROGENATION WORKERS* EXAMINED FOR FIVE YEARS

Histological Diagnoses	Number of Cases	Mean Subject's Age	Length of Exposure (months)	
			Min.	Max.
Pitch Acne[1]	3	30	10	74
Chondrodermatitis[1] helicis	3	33	3.5	52
Keratoses[2]	17	39	10	116
Keratoses	8	44	17	96
Acanthoses and hyperkeratoses	9	40	5	108

* White Males Only

1. Clinical Diagnoses Only

2. Diagnoses by a single pathologist only

SOURCE: Sexton (34) and SRI International (44).

TABLE 10

MAJOR CONSTITUENTS OF COAL CONVERSION PROCESS STREAMS AND EMISSIONS

CONSTITUENTS	PHYSICAL STATE	OSHA STANDARD*	OCCUPATIONAL SIGNIFICANCE
ALIPHATIC HYDROCARBONS	GAS OR VAPOR		UNLIKELY THAT MOST WILL PRESENT SIGNIFICANT HAZARD, EXCEPT FOR DODECANE - A POTENTIATOR OF SKIN TUMORIGENESIS BY BaP
AMMONIA	GAS	50 PPM	NO EVIDENCE OF ILL EFFECTS FROM PROLONGED EXPOSURE TO SUBIRRITANT CONCENTRATIONS
AROMATIC AMINES	LIQUIDS OR SOLIDS	5 PPM FOR ANILINE AND TOLUIDENE; NO SAFE LIMIT FOR B-NAPTHYLAMINE AND BENZIDENE	ANILINE & SUBSTITUTED BENZENES ARE HIGHLY TOXIC; B-NAPTHYLAMINE AND BENZIDENE ARE POTENT CARCINOGENS
SINGLE-RING AROMATICS	LIQUIDS AND SOLIDS	10 PPM FOR BENZENE	VAPOR HAZARDS ARE ONLY LIKELY WITH BENZENE AND RELATED COMPOUNDS OF RELATIVELY LOW MOLECULAR WEIGHT
CARBON DISULFIDE	LIQUID	20 PPM	ACUTE EFFECTS WOULD ONLY BE RESULT OF LARGE LEAK IN PROCESS STREAM; CHRONIC POISONING IS MORE SERIOUS HAZARD
CARBON MONOXIDE	GAS	50 PPM	CHRONIC LOW-LEVEL EXPOSURE EFFECTS ARE CONTROVERSIAL
CARBONYL SULFIDE	GAS	NO STANDARD	LITTLE EVIDENCE OF HUMAN TOXICITY ALTHOUGH IT IS PROBABLY LESS HAZARDOUS THAN H_2S
HETEROCYCLIC AROMATICS	LIQUIDS AND SOLIDS	5 PPM FOR PYRIDINE; NO STANDARD FOR ACRIDINE	N-HETEROCYCLIC COMPOUNDS ARE THE ONES OF MAIN INTEREST (SKIN AND MUCOUS MEMBRANE IRRITANTS, POSSIBLE POTENTIATORS
HYDROGEN CHLORIDE	GAS	5 PPM CEILING	IRRITANT

TABLE 10 (CONT.)

CONSTITUENTS	PHYSICAL STATE	OSHA STANDARD*	OCCUPATIONAL SIGNIFICANCE
HYDROGEN SULFIDE	GAS	20 PPM CEILING; 50 PPM 10 MIN. PEAK	STRONG IRRITANT; POSSIBLE COAGENT FOR CARCINOGENS
MINERAL DUST & ASH	PARTICULATE	--	POSSIBLE VEHICLE FOR PAH
NICKEL CARBONYL	LIQUID	$7 ug\ m^{-3}$	VAPOR INHALATION AFFECTS CNS & MAY INDUCE CHEMICAL PNEUMONITIS
NITROGEN OXIDES	GASES	5 PPM FOR N-DIOXIDE	SOURCE OF NO_x (SEE NEXT LINE)
NITROSAMINES	LIQUIDS & VAPORS	EXCEPTIONALLY DANGEROUS CARCINOGEN	IT HAS BEEN HYPOTHESIZED THAT NO_x FROM COMBUSTION MAY REACT WITH VAPOR FROM GLYCOLAMINE SCRUBBERS TO YIELD THESE CARCINOGENS.
PHENOLS	SOLIDS OR LIQUID MIXTURES	5 PPM	POTENTIAL ENHANCEMENT OF SKIN AND RESPIRATORY CARCINOGENS
POLYCYCLIC AROMATICS	SOLIDS	NO SAFE LIMITS	WELL ESTABLISHED AS SKIN CARCINOGENS, LESS SO AS RESPIRATORY CARCINOGENS. BaP IS HIGHLY CARCINOGENIC
TRACE ELEMENTS - As, Be, Cd, Pb, Mn, Hg, Se, and Va	SOLIDS	2(Be) - 500 (As, Va) $ug\ m^{-3}$	PROBABLY NO ATMOSPHERIC HAZARD IN VICINITY OF PLANT, BUT THEY MAY POSE HAZARD TO WORKERS CLEANING FILTERS & STACK DEPOSITS AND MAINTAINING TOPS OF GASIFIERS
SULFUR OXIDES	GAS OR AEROSOL	5 PPM	POSSIBLE COCARCINOGEN

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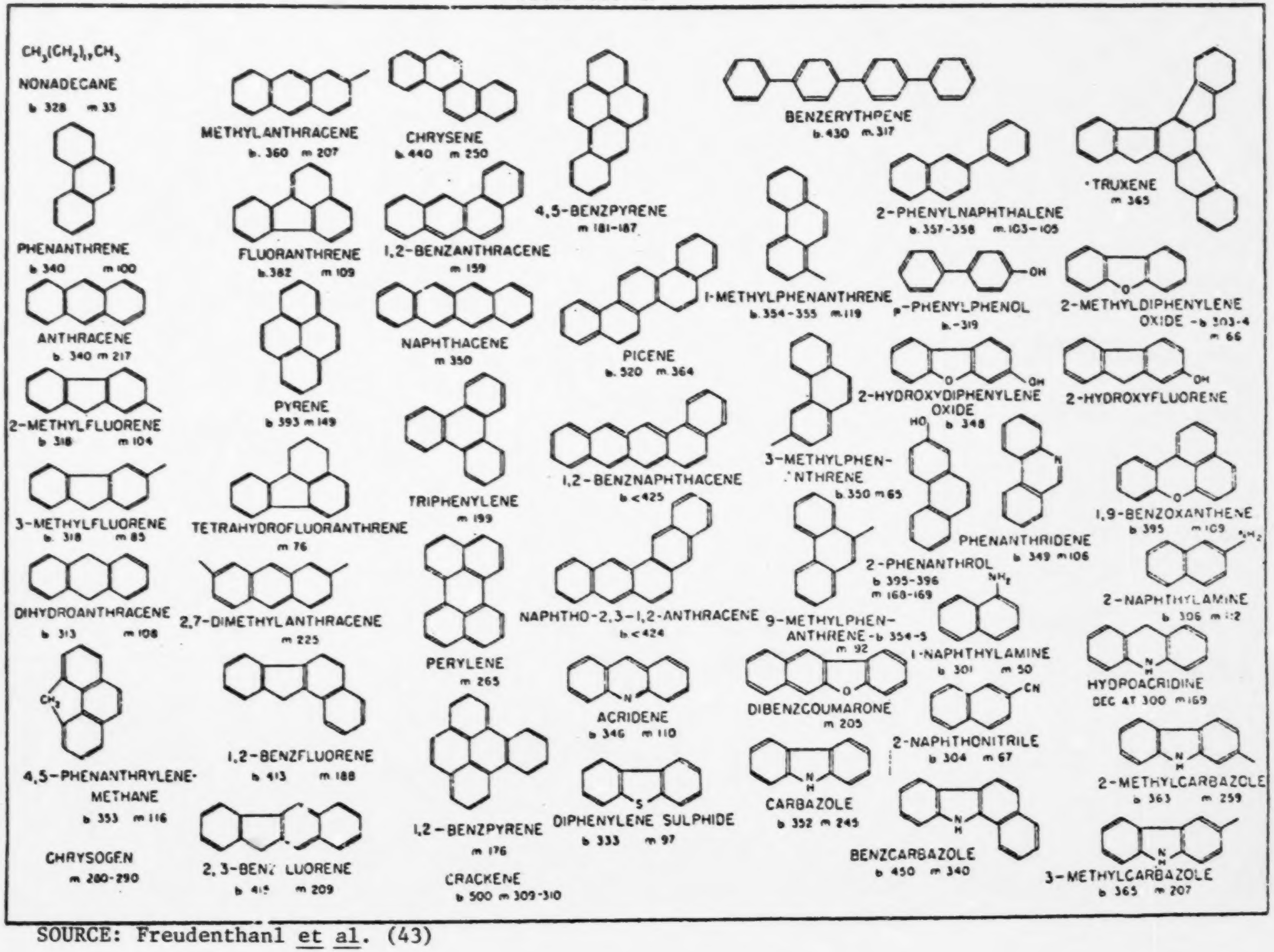
*- TIME-WEIGHTED AVERAGE UNLESS OTHERWISE SPECIFIED

ADAPTED FROM ENVIRO CONTROL (42)

FIGURE 1

POLYCYCLIC AROMATIC CONSTITUENTS OF COAL-TAR
BOILING POINT RANGE
ABOVE 300° C.



SOURCE: Freudenthanl et al. (43)

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LONG TERM HEALTH IMPLICATIONS OF RADIOACTIVE WASTE DISPOSAL

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The Long Term Health Implications of Radioactive Waste Disposal

Introduction

Any projection into the future is an extropolation based upon experience and models or clairvoyance. In the absence of reliable application of the latter, projections will be no better than the models used. Thus any discussion of the long term health impact of radioactive wastes depends upon a sequence of models from source terms to exposed populations and their capabilities.

The time interval of interest is that after institutional controls are no longer expected to be maintained and longer. That is, about 100 years to 10,000 or 100,000 years from the present. Models for projections over such time periods depend on major assumptions regarding the size and nature of future societies as well as estimates of the future behavior of nature and ecological systems. Thus the uncertainties in such projections will be large; the question of whether the uncertainties are critical can only be ascertained after analysis is complete. The uncertainty of critical parameters will, therefore, be dominant; but a limit analysis, i.e., estimation of minimum, best estimate, and maximum ranges can be very informative and useful. An attempt at this is made here.

Models for Estimation

The array of models used for estimation of long term impact begins with estimation of source terms for all forms of waste, i.e. high level and transuranic, mill tailings, and other wastes. High level wastes consists of fission products from fuels reprocessing and unreprocessed spent fuel, or any other materials whose activity level is high enough to cause acute effects with only ordinary precautions. Transuranic elements are considered as a part of the high level waste problem since similar disposal methods may be used. Mill tailings are the residues from the milling of uranium and thorium. Other wastes are all those whose effects are primarily chronic with ordinary precautions, and includes such things as "low level wastes," material and rubble from decomissioning, or any thing else. Table 1 characterizes these by their volume and activity levels.

The second model involves the pathways to the environment over time. There are three parts, 1) the selection of a disposal method and site with specified design objectives, 2) pathways to the environment allowed by the design, 3) and pathway to the environment from external events caused by man or nature. A third model is required to convert exposure to health effects for individuals and generic populations, and finally a fourth model is required to specify the size and behavior of future populations and societies.

Each model is inter-related to assumptions in the others and cannot be developed without such recognition. Table 2 summarizes these models, the details will be covered when the models are applied.

Source Terms

The long term impact of radioactive waste disposal must be measured after repositories are sealed or institutional controls ended. Further, the impact of the total practice must be ascertained, not that of a single repository. For energy production by fission the amount of waste generated for a typical 1000 MWe*/yr plant is summarized in Table 3.More detailed estimates are available such as those by Blomeke and Lee(1),but are not required for the limit analysis attempted here. OECO and UNSCEAR estimates based upon uranium reserves at reasonable cost, that about 10,000 GWe years are available from fission energy. This may be increased by a factor of about three for use of thorium and higher priced uranium. Use of breeder reactors can extend this to about a million GWe years to a first approximation. Thus, the total practice for a world population of 10 Billion (2 1/2 times our present population) represents one kilowatt per person, not per year, but totally, for uranium fission. This is equivalent to 2 watt-years per year for 500 years. For breeders this goes up to 200 watt-years per year for 500 years.

Table 4 estimates the total wastes from these practices. They provide a first cut for describing a total health impact limit. However, they can only be used with more detailed estimates. So far only one estimate of health impact has been made for advanced disposal practices for exposure at a significant level of detail. This is the Environmental Protection Agency analysis of impact from high level wastes for the purpose of establishing regulatory standards. This analysis is based upon a base line, deep geological repository with no planned releases, and forms the basis for health impact estimates made here.

High Level Waste Analysis

The objective of the EPA study was the synthesis of the probabilities and consequences all significant events that might occur over a substantial period of time. The results shown here are from congressional testimony given by Dan Egan(2).

The analysis is based upon a model deep geologic repository with no planned releases over its lifetime. Impact will depend upon the designed repository to perform not as planned or disturbances by external forces. Thus, only impacts from "unplanned" inadequacies in repository design or "accidental" disruptive events have been addressed. Only impacts which might occur after final backfilling and sealing of the repository have been investigated. Sealing of the repository is established as the initial point of the time scale in which the potential impacts are considered. The analysis addresses total amounts of radioactivity which might be released to the environment over various long time periods after repository sealing. Most attention has been devoted to calculating total impacts incurred over the first 10,000 years after sealing. Radionuclide concentrations in various geologic or environmental pathways are generally not calculated. Thus, this analysis is not directly relevant to determination of radiation doses or health risks to individual members of a population.

"Definition of a baseline geologic setting. Initially this has been chosen as a bedded salt repository, with over- and underlying aquicludes (relatively impermeable strata) immediately adjacent to the repository strata, and aquifers on the other side of both aquicludes.

Definition of a baseline repository design. The details of the baseline repository design selected for analysis are shown in Table 5. The repository was assumed to be a spent unreprocessed fuel repository containing about 100,000 metric tons of heavy metal (MTHM). It was also assumed that all the spent fuel would have aged for approximately ten years after reactor discharge before the repository was sealed " (2).

Sequences of events or process which could initiate releases of radionuclides were identified by an extensive study undertaken for EPA by Arthur D. Little, Inc. The events were identified and the range of the rate of occurrence for each event were made. Upper bound estimates and more realistic lower value estimates were made for some 60 different sequences of events and are categorized as shown in Table 6.

For releases to the air or land surface, the fraction is the portion of the repository inventory directly expelled. For releases to ground water pathways, the fraction represents the portion of the inventory subjected to contact with, and potential transport by, ground water. In addition, for releases to ground water pathways, either the permeability of the flow path resulting from the event is estimated or a water velocity through the affected area was provided. A simple one-dimensional ground water transport model, neglecting axial dispersion, was employed to approximate the flow of radionuclides from the repository to surface water bodies. This model was determined to be adequate, compared to more sophisticated techniques, for the purpose of calculating total radioactivity reaching the environment over long periods of time. Figure 1 depicts the geometry employed in the model. Table 7 indicates the various parameters used in the calculation, including the retardation factors initially assigned to the various radionuclides.

The radioactive releases have been translated to health effects incurred by an assumed average population. Person-rems to the various organs were calculated using average pathway and population models developed by the EPA staff. These were then converted to health effects using the 1972 BEIR estimates(4).These models are intended as representations of pathways as they are currently understood. The health impacts indicated are not intended as predictions of future effects, unless future pathways and populations are assumed to be reasonably similar to those of today.

In order to develop probability and consequence data a set of hypothesized event times was selected, based upon establishing appropriate "event time periods." The occurrence rate for each event chain was then integrated over this event time period at arrive at a probability of that event occurring within the period, with the specific event time being taken as the midpoint of the period.

Consequences are developed with respect to the time over which the environmental impact is being considered. For releases which involve sudden removal of material from the repository (releases to either air or land surface) the consequences are assumed to occur at the event time. Releases through ground water pathways would be drawn out over time due both to the slowness of the ground water transport and the dissolution of the radioactive waste. Dose commitments over the time period of interest are then calculated.

Using the techniques outlined above, probability/ consequence data points are generated. These data have been summarized in two ways. First, the sum of the products of these data points is calculated. This result, which is variously called the "expected value" of the environmental impact, or the "risk", is an estimator of the health effects "expected" within the dose commitment period of interest. Second, the probability/ consequence points are depicted graphically through the use of a "cumulative complementary distribution function (ccdf)". Figure 2. indicates the two methods used to summarize the analysis.

"Figures 3 and 4 depict the format of the results obtained by applying the above methodology to the "high value" and "low value " occurrence rates provided by A. D. Little. A primary purpose of this presentation is to focus upon the format of this analytic methodology and its potential usefulness for environmental standards, as opposed to the numerical values off preliminary results which are still being extensively reviewed. As a tool to encourage this, several of the numerical parameters have been deleted from Figures 4 and 5. However, it can be noted that the sum of the products of probability and health effects (which will be termed the "expected value" risk) for the 10,000 year period has been in the range of 100-1000 for the "high value" rates, with the "low value" rate results being about four orders of magnitudes smaller"(2).

There are several observations which can be drawn from Figure 3. First, over this 10,000 year period, with the "high value" event chain rates estimated by A. D. Little, it is virtually certain that some health effects will be incurred from radionuclide releases. Thus, although no "planned" releases are forecast, "unplanned" events or "accidents" are expected sufficiently often that releases up to a certain level could still be projected with a probability approximating one. As Figure 4 indicates, the situation relative to the "low value" rates is quite different"(2).

Further, the bulk of the "expected value" risk is attributable to ground water related pathways.

"This "expected value" risk can also be subdivided according to the radionuclide causing the impact, and preliminary results of such analyses are indicated in Table 8.

The dominance of technetium-99 is surprising only until one considers its relatively high fission yield (on a gram-atom basis), its long half-life (213,000 years), and its assumed lack of sorption in geologic media. Many radionuclides which have often been considered more characteristic of the hazards of high-level waste (strontium-90, cesium-137, plutonium-239, etc.) do not appear to be significant in this analysis because either their short half-life (strontium-90, cesium-137) or their assumed strong sorption in geologic media (plutonium and other actinides) prevent them from reaching the accessible environment through ground water pathways within 10,000 years(4).

The base line system used by EPA will accommodate 100,000 MTHM as shown in Table 5. Three such repositories of this type would suffice for disposal of the total high level waste of 10,000 GWe-years of uranium fuel cycle operation. (See first row and column of Table 4.) This would mean an upper limit of 3000 health effects for the total cycle and about 0.3 for the most optimistic estimate. Table 9 summarizes this data.

In order to put these risks in perspective they may be compared to a number of bench marks: which are shown in Table 10. Even the upper level of risks from waste disposal (0.3 per year for 10,000 years) is small compared to the benchmarks.

Low Level Wastes

There have been no extensive studies conducted on the long term impact of disposal of low level wastes or mill tailings to the extent carried out by EPA for high level waste disposal. Thus, only a limit analysis can be made for low level and mill tailings wastes.

The IRG report indicates that there are 16 x 10^6 ft^3 of low level waste at the six commercial sites in the United States. About 35 times this volume of waste would need to be disposed of under current schemes for 10,000 GWe years of operation although advanced volume reduction would reduce this by a factor of 10. Greater amounts are required for expanded fuel cycles.

Some of six commercial sites have been closed down because releases to the environment have been greater than expected. Thus some level of "planned" release may exist if present methods are used for low-level disposal. More important after institutional control lapses these sites are subject to internal design failures and a whole array of external factors, including erosion and human intrusion, which are not as important in deep geological disposal. This is also true for mill tailings disposal if by present practices as well.

Without evaluation of all of these factors for different disposal methods, no health impact evaluation is possible. What I have chosen to do instead is to use Blomeke and Lee's (1) estimates of the volume of air for dilution of a given volume of material to concentrations specified in DOE (ERDA) concentration guides as a measure possible impact, and then to determine the decontamination (or retention) factor, d.f., needed to bring the impact into the same ball park as those for high level waste disposal. Thus, the "d.f." will be a measure of the "effectiveness of containment" needed for a repository.

The total mass of the atmosphere has been estimated at 5.2×10^{21} gms.(5)with a density of .00122 gms/m^3 at sea level. This would indicate that a total volume of air at about $4 \times 10^{24} m^3$. Blomeke and Lee have used dilution factors as follows.**

Non Transuranic Low Level Wastes	$1 \times 10^9 m^3$ of air/m^3 waste
Mill Tailings	$1.6 \times 10^{10} m^3$ of air/m^3 waste

For 10,000 GWe years, about $5 \times 10^8 m^3$ of low waste disposal and $8 \times 10^{14} m^3$ for mill tailings accumulate for the uranium fuel cycle (Table 4). For dilution this would require $5 \times 10^{17} m^3$ of air for low level waste and $1.3 \times 10^{25} m^3$ of air for mill tailings. For breeders $5 \times 10^{19} m^3$ is required for low level waste while tailings stay about the same. The mill tailings dilution volume exceed that available in atmosphere just to meet DOE standards. Thus a retention factor of three is required for mill tailings just to keep it at DOE maximum levels. Mpc value for DOE standards are based upon concentration guides of 500 mrem/yr to an individual, and exposure to 10^{10} people of this level is 5 x 10^9 man rems/yr or about 10^6 health effects per year based upon ICRP models. This is a factor of 3.3 x 10^6 higher than the upper limit estimate for the fuel cycle in Table 9. To achieve these levels a retention factor of about 10^7 per year is required for mill tailings over their lifetime. Low level waste retention cannot be adduced in this way.

Thus in summary the retention factor for mill tailings indicates that this may be the key disposal problem. Whether the d.f. level can be achieved has yet to be determined. It may be quite easy to do so, but at least this provides an initial target for disposal retention capability. Retention requirements for low level wastes may have to consider individual exposures as a limitation.

This paper does not give definitive answers. There are none. But high level wastes have limited health impact. Low level waste impact has yet to be determined. Mill tailings may be the real problem for waste disposal.

TABLE 1

Some Generic Waste Classifications

Waste Type	Relative Volume	Exposure Activity
High Level	Small	Acute
Fission products from Reprocessing	"	"
Spent Fuel Rods	"	"
Discrete Sources	"	"
Transurancies	Small to Medium	Chronic
Mill Tailings	Large	Chronic
Other Wastes		
Low Level Waste	Medium	Chronic
Rubble, etc.	Medium to Large	"

TABLE 2

Models Required for Health Impact Estimation

1. Source Terms
   - High Level - spent fuel or reprocessed
   - Low Level
   - Tailings and diffuse wastes

2. Pathways to the Environment
   - Disposal Method and Site
   - Designed Pathways
   - Unplanned Events

3. Exposure Health/Effect Determination
   - Dose Estimation - Internal and External
   - Population Commitments
   - Individual Doses

4. Future Population Model
   - Size and Growth
   - Protective Capability

TABLE 3

ANNUAL WASTE GENERATION RATES (3)
(Normalized to an Average 1000 MWe
Light Water Reactor)

Spent Fuel Discharged	25.4 MTHM/yr (332 ft^3/yr)
Low Level Waste, Generated Onsite 1/	
a) Present Experience	45,000 ft^3/yr
b) Design Basis	15,000 ft^3/yr
c) Advanced Volume Reduction 2/	5,000 ft^3/yr
Low Level Waste, Generated Offsite	
a) Uranium Mill, Tailings Solutions 3/	254,000 MT/yr
Tailings Solids 3/	96,000 MT/yr
b) UF$_3$ Conversion	1,200 ft^3/yr
c) Enrichment 3/, 4/	50 ft^3/yr
d) Fuel Fabrication	750 ft^3/yr
Transuranic wastes, Generated Onsite and Offsite	0

NOTE: MT = Metric Tons
HM = Heavy Metals

1/ Roughly 40% of current volumes generated is contaminated trash.

2/ This estimate reflects the use of methods which are presently not economical Current, allowable activity levels per package may preclude actual achievement of this level in the future.

3/ These wastes are currently disposed of at the processing facility sites.

4/ This value is based on gaseous diffusion technology. The new centrifuge process could potentially generate more (up to 2900 ft^3/yr.

TABLE 4

SOME ROUGH ESTIMATES OF THE TOTAL RADIOACTIVE WASTES FROM FISSION ENERGY PRODUCTION

Type of Waste	AMOUNT PRODUCED			
	Uranium 10^4GWe-yrs.	Uranium & Thorium $3x10^4$GWe-yrs.	Breeders 10^6GWe-yrs.	Units
Spent Fuel	$3x10^6$ $3x10^5$	$1x10^7$ $1x10^6$	$3x10^8$ $3x10^7$	ft^3 MT(HM)
Low Level Wastes-on site				
A. Present Practice	$5x10^8$	$2x10^9$	$5x10^{10}$	ft^3
B. Design Basis	$2x10^8$	$6x10^8$	$2x10^{10}$	ft^3
C. Advanced Volume Reduction	$5x10^7$	$2x10^8$	$5x10^9$	ft^3
Low Level Wastes-off site				
A. Uranium (Thorium) Tailings				
Solutions	{$3x10^9$ $6x10^{14}$}	$1x10^{10}$ $2x10^{15}$}	$3x10^{11}$ $6x10^{16}$}	MT ft^3
Solids	{$1x10^9$ $2x10^{14}$}	$3x10^9$ $6x10^{14}$}	$1x10^{11}$ $2x10^{16}$}	MT ft^3
B. UF Conversion	$1x10^7$	$2x10^7$		ft^3
C. Enrichment	$5x10^5$	$1x10^6$		ft^3
D. Fuel Fabrication	$1x10^7$	$3x10^7$	$1x10^9$	ft^3

TABLE 5. PARAMETERS FOR BASELINE REPOSITORY (2)

	High-Level Solidified Waste	Spent Fuel
Depth of Repository Horizon (feet)	1,500	1,500
Mined Area (acres)	2,000	3,500
Capacity (canisters)	35,000	320,000
Total metric tons heavy metal (MTHM) charged to reactor	100,000	100,000
Specific Heat Input (kw/acre) - maximum	150	60

TABLE 6. CATEGORIZATION SYSTEM FOR EVENT CHAINS AND SOME TYPICAL EVENT CHAIN PARAMETERS (2)

Event	"High Value" Occurrence Rates (per year)	"Low Value" Occurrence Rates (per year)	Fraction of Repository Involved
DIRECT ATMOSPHERIC RELEASES (AIR)			
Meteorite Impact	3×10^{-10}	3×10^{-11}	0.20
DIRECT RELEASES TO LAND SURFACE (LS)			
Gas/Oil Exploration	0 for 0-100 yrs 9×10^{-6} after 100 years	always 0	0.00002
CONVECTIVE RELEASES TO AQUIFER (AQ)			
Seismicity/Faulting	ranges from 2×10^{-6} to 4×10^{-8}	ranges from 4×10^{-11} to 1×10^{-13}	0.01
CONVECTIVE RELEASES DIRECT TO SURFACE WATER (SW)			
Breccia Pipes	ranges from 1×10^{-4} to 2×10^{-9}	ranges from 5×10^{-10} to 8×10^{-15}	0.002

TABLE 7. - PARAMETERS USED IN GROUND WATER PATHWAY CALCULATIONS (2)

Permeability along Aquifer or through fractured overburden:		1×10^{-4} cm/sec
Flow Gradient along Aquifer:		0.1
Flow Gradient through fractured overburden:		0.01
Retardation Factors for Significant Radionuclides:	C - 14	10
	SR- 90	100
	CS-137,135	1000
	I -129	1
	TC- 99	1
	NP-237	100
	PU-238,239,242	10000
	AM-241	10000
	CU-244	3300

TABLE 8

- BREAKDOWN OF "EXPECTED VALUE" RISK BY RADIONUCLIDE (2)

Radionuclide	Per cent of Impact
TC- 99	88
C - 14	7
AM-241	3
I -129	1
SR- 90	less than 1
PU-239	less than 1
remaining radionuclides	much less than 1

TABLE 9

RANGES OF HEALTH EFFECTS FROM HIGH LEVEL RADIOACTIVE WASTE DISPOSAL FOR THE TOTAL FISSION ENERGY PRACTICE

	1 Repository 10^5 MTHM	Uranium Cycle $3x10^5$ MTHM	Uranium & Thorium Cycles $6x10^5$ MTHM	Breeders $3x10^7$ MTHM
Upper Limit Health Effect	1000	3000	9000	100,000
Lower Limit Health Effects	0.1	0.3	0.9	10

TABLE 10

Some Radiation Risk Benchmarks
(Based upon EPA calculations)(3)

Risks From Nuclear Weapons Fallout (Tritium, Cesium-137, Carbon-14, Plutonium)	700 health effects/yr.
One Percent of Natural Background	100 health effects/yr.
Risks From Undisturbed Natural Uranium Ore Bodies	10-15 health effects/yr.
Risks From Nuclear Energy Operation (10,000 GWe years based upon 40 CFR 190)	200 health effects/yr. (First 100 years)
	30-60 health effects/yr. (Next 10,000 years)

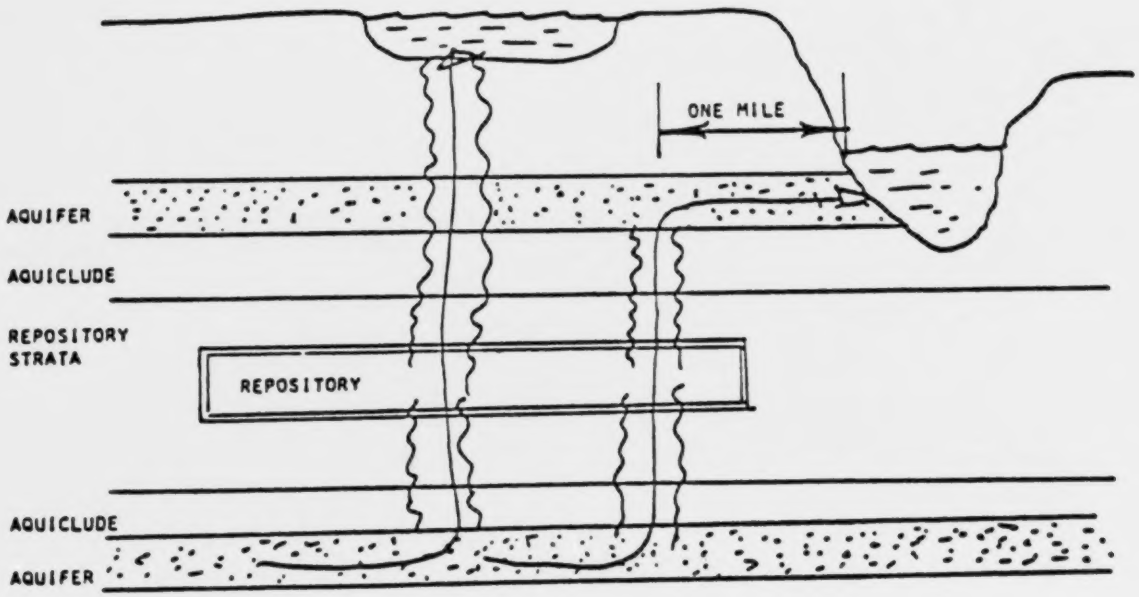


FIGURE 1. Schematic of Ground Water Flows (2)

THE PROBABILITY AND CONSEQUENCE CALCULATED FOR EACH SUCH EVENT SCENARIO ARE THEN COMBINED AND EXPRESSED IN TWO WAYS: (2)

'EXPECTED VALUE' -- $\sum_i (P_i)(C_i)$

'COMPLEMENTARY CUMULATIVE DISTRIBUTION FUNCTION (CCDF)' --

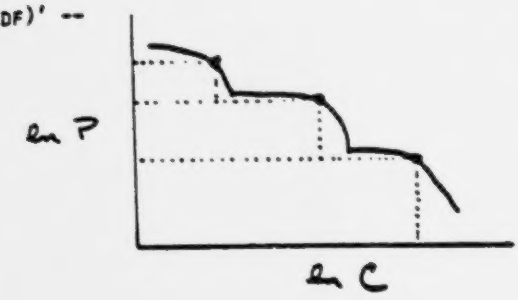
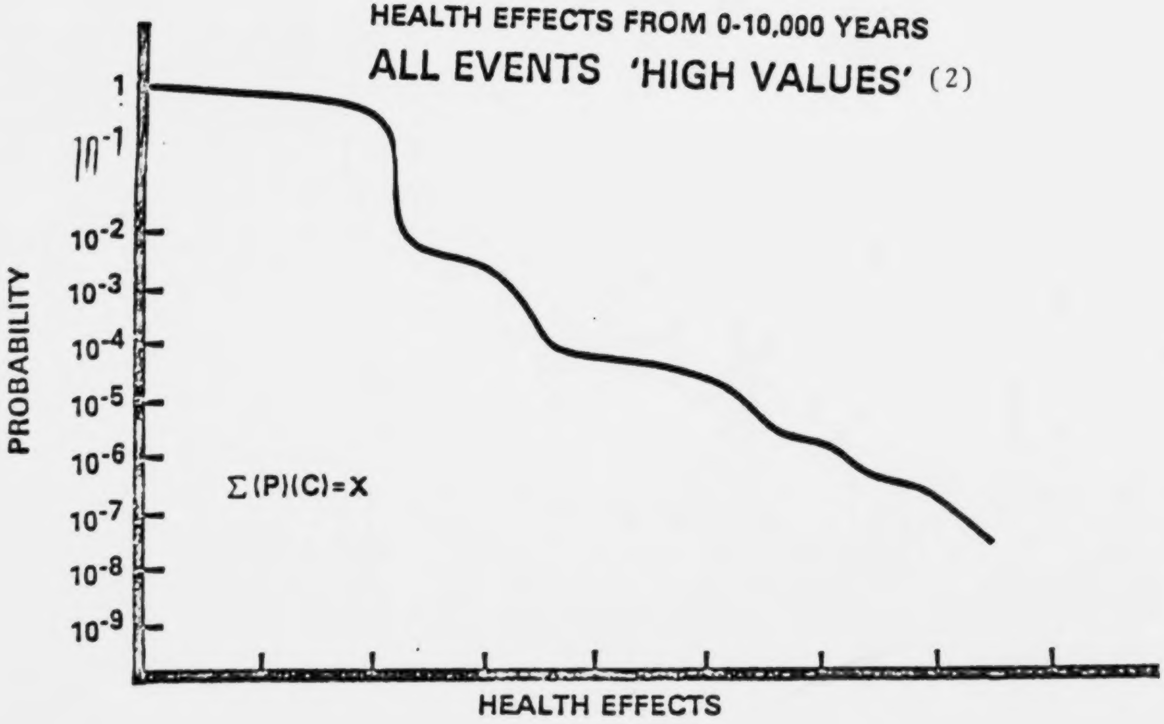


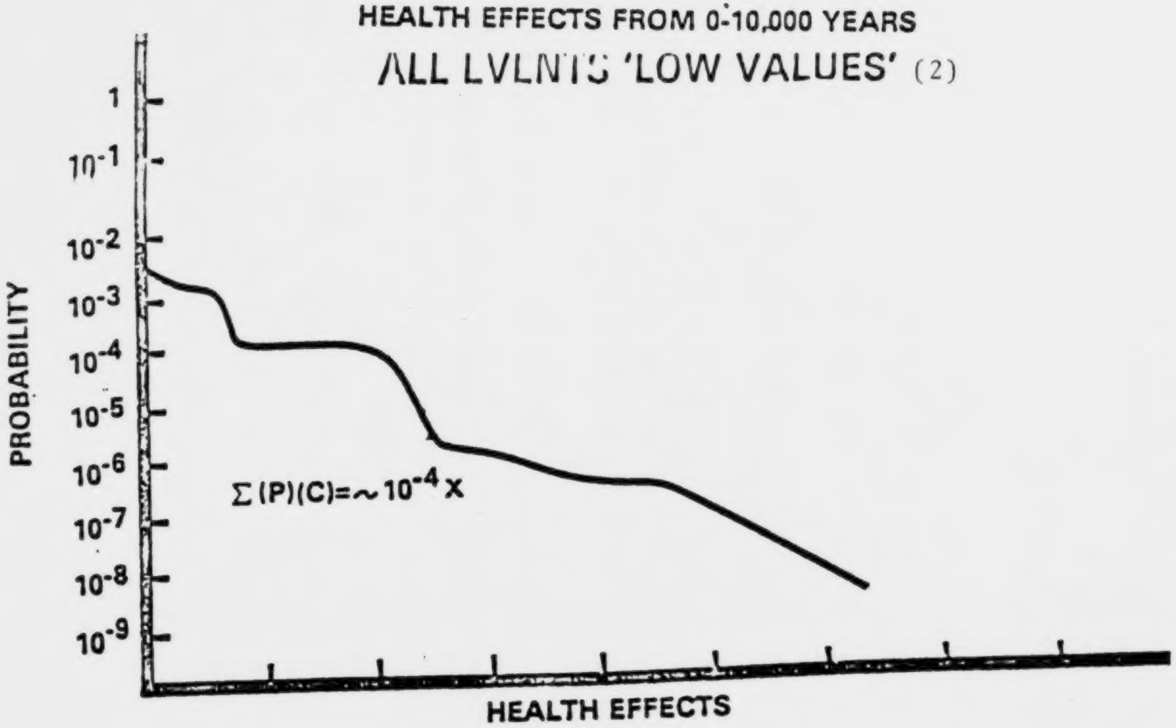
FIGURE 2



HEALTH EFFECTS FROM 0-10,000 YEARS

ALL EVENTS 'HIGH VALUES' (2)

FIGURE 3



HEALTH EFFECTS FROM 0-10,000 YEARS
ALL EVENTS 'LOW VALUES' (2)

FIGURE 4

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*Calculated from Tables 11 and 12 of Blomeke and Lee, (1).

AREAS OF UNCERTAINTY IN ESTIMATES OF HEALTH RISKS

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INTRODUCTION

Confusion is engendered in estimates of energy-entailed health risks by the misundertstanding between objective measures of risk - which describe the estimated real or actual risk of a process-and the subjective perception of these risks; the subjective perception of risks colors much of the thinking of most decision-makers and the public. The situation is worsened by propaganda from special-interest groups. In assessing health risks from energy production and use, one must map where are the gaps in knowledge, yet necessarily basing the assessment on state-of-the-art- information.

Another uncertainty is engendered by confusion as to how health assessment enters into energy assessment. Estimates of health risks and their costs are an essential in energy-policy along with (a) direct costs of energy production; (b) other costs generated in a way similar to the assessment of health costs; and (c) other politico-societal considerations. Figure 1 diagrams the position of biomedical assessment in overall energy assessment. The demand for energy by the consumer is the starting point. Nationally or regionally, this demand can be met by varying combinations of existing energy-generating systems. Or, if assessment of research on new energy systems is at the forefront of interest and necessity, one must foretell how best to mix existing energy-generating systems and alternatives arising from research and development.

To measure the real health costs of various energy sources, one needs to put them in the framework of logical, comprehensive energy analysis. Accordingly, Figure 2 diagrams stages in energy production, distribution and use; it permits coherent analysis of costs and hazards at each state. The diagram includes: (1) hydropower; (2) nuclear fuel; (3) coal; (4) oil; (5) natural gas and (6) new technologies to be developed. The steps in the fuel cycles comprise: (1) exploration and extraction; (2) refining and conversion; (3) transport; (4) central station conversion; (5) transmission and distribution; (6)

decentralized conversion; (7) conversion by final energy users. Most of these process steps entail unique biomedical, environmental, and other costs, some direct (e.g., risks of injury or death in mining), some indirect, (e.g., release of pollutants into air, water, thence into food chains, etc.).

Such analysis helps: (1) to evaluate alternative energy policies with due reference to health costs; (2) determine where money must be spent to reduce health costs; and (3) decide where to allocate research and development funds for determining health and environmental costs.

This paper is concerned with several currently prominent uncertainties in estimating health risks from electric-power generation: (1) health effects of air pollution, especially acid sulfates and respirable particulates; and (2) low-level radiation effects. With these uncertainties in mind, state-of-the- art current assessments of various fuel cycles on a unit plant basis are given and a method outlined for using these results in national or regional energy assessment.

HEALTH EFFECTS OF AIR POLLUTION

Coal combustion produces a wide range of air pollutants, including particulates -- SO_2, NO_x, CO; polycyclic aromatic hydrocarbons (PAH); and trace metals, e.g., iron, mercury, and cadmium. These primary pollutants contribute to atmospheric chemical reactions producing secondary pollutants such as ozone, sulfate, and nitrates. Because exposures to many pollutants are simultaneous, it is difficult to assign damage by air pollution to individual agents. Considerable evidence links sulfur-particulate air pollution with health damage.[1-3] It is currently hypothesized that the causative agents are sulfate compounds produced by oxidation of SO_2 in the atmosphere. Table 1 summarizes the mechanisms that convert SO_2 to sulfates. As seen, oxidation of SO_2 to sulfates depends on the presence of other pollutants such as NO_x, oxidants, heavy metal ions, and particulates.

There is much uncertainty in understanding the mechanisms by which SO_2 is oxidized to sulfate: the role of heterogeneous transformation processes (SO_2 oxidation, SO_2-O_3-liquid-water interaction, SO_2-H_2O_2-liquid-water interactions, SO_2 oxidation of graphitic materials and other aerosols, etc.); aerosol transformation (nucleation growth, gas-aerosol reactions); and SO_2-free-radical reactions (OH, HO_2, organic radicals) all need research. The fact that presence of other pollutants determines the formation rate and the final form of sulfate is important in underscoring the difficulty in ascribing the health effects of air pollution to individual agents.

Particulate emissions from combustion are today largely controlled by mechanical devices, e.g., cyclone, electrostatic precipitators, and, occasionally, fabric filtration. The chemical composition of emitted aerosols (particulates), especially surface composition, depends on their chemical history and is poorly understood; the smaller sizes present disproportionately more surface, and thus serve as an adsorption site for effluent species in post-combustion gases. An important consideration is that even after removal of most of the particulates, the oxidation of SO_2 to sulfates in the atmosphere results in the formation of secondary particulates in the respirable range ($<2\mu m$ down to submicrometer range). These persist longest in the atmosphere and penetrate most into human airways since particle size is an important variable that affects the site of deposition of sulfur oxides in the respiratory tract and the size of the response. Figure 3 diagrams the effect of particle size on deposition of particles in the respiratory tract. Small particles are deposited deeper in the respiratory tract than large particles.

Support for the hypothesis linking sulfur-particulate air pollution with health damage comes from many sources, the most dramatic from the major, amply documented episodes of air pollution -- Meuse Valley in Belgium; Donora, Pennsylvania; and London -- which clearly showed that air pollution, when severe, caused widespread illness and death.

One of the worst air pollution disasters was in December 1952. A dense, cold fog settled on London for four days, suddenly increasing deaths. Excess deaths during the fog or shortly afterwards were estimated at 2,500-4,000 in greater London (population 8.3 million). Table 2 gives figures for the smaller area of the county of London. Deaths from bronchitis contributed most to the rise in deaths. Deaths from other diseases with impaired respiratory function also increased. There were increased deaths from heart disease, presumably due to strain from impaired respiratory function or to a direct effect. Death from other causes also increased; this residual mortality was significant; it is unlikely that it was due to respiratory impairment. The increases in mortality in London in 1952 were correlated with vast increases in smoke shade and SO_2, measured at the same time. Concentrations of smoke shade were too great to be measured accurately; the 48-hour average of ~ 4.5 mg/m^3 at a central site is thus a conservative estimate. Concentrations of SO_2 were as high as 3.7 mg/m^3 (48-hour average). Smoke shade and SO_2 were monitored in the United Kingdom since 1912; their choice reflected the view that these were the important pollutants.

Figure 4 relates the incidence of chronic bronchitis to SO_x precipitation for several Japanese cities.[4] There is a linear correlation between chronic bronchitis and SO_x precipitation present even in the absence of smoking. Smoking 11-20 cigarettes/day (curve a) or 1-10 cigarettes/day (curve b) clearly exacerbates the effects of the SO_x.

Figure 5 plots deaths in Oslo, Norway against weekly SO_2 concentration.[5] The relationship is linear. Since we know that SO_2 by itself does not kill or cause chronic bronchitis, it is probably a chemical transformation product of the SO_2 that is the crucial factor. The fact that Britons, Japanese, and Norwegians succumb to damage from air pollution indicates the absence of specific ethnic resistance. This is amply confirmed by results from the United States Environmental Protection Agency's CHESS (Community Health and Environmental Surveillance System) studies.[6]

Figures 6-8 give examples of the results of the CHESS studies. Figure 6 plots the percent excess acute lower respiratory disease in children against annual average suspended sulfates concentration ($\mu g/m^3$) (studies in six areas). Figure 7 plots aggravation of heart and lung disease in the elderly (given as percent excess) against the 24-hour suspended sulfates concentration

($\mu g/m^3$) (studies in two areas). Figure 8 plots the percent excess mortality rate in New York City in the 1960's against the 24-hour suspended sulfates concentration ($\mu g/m^3$). For comparison, the figure includes data on excess mortality in London in the 1950's and Oslo in the 1960's.

Although many have criticized these epidemiological studies and their pollution measurements, and because of the uncertainties emphasized by these criticisms, one cannot use the results of the CHESS studies to estimate dose-effect relationships. Yet, despite all their limitations, the CHESS studies provide weighty evidence that "sulfates" -- or something closely related to them -- damage health. The data are compatible with the notion that the more sulfate, the more damage.

A vast literature associates air pollution (smoke shade, total suspended particulates, and SO_x) with sickness and death.[1-3] Again, since one now knows that it is not the SO_2 which damages it is probably some chemical transformant of SO_2 that is responsible: this is the material that forms a respirable particulate that probably constitutes most of the smoke shade and most of the noxious material in the respirable fraction of total suspended particulates. This body of evidence supports the hypothesis that sulfates as respirable particulates or something closely associated with them does the damage.

Impressive evidence for damage by sulfates has come from laboratory studies in animals. The acid sulfates proved the more irritating and toxicity was also related to particle size (Table 3).[8,9]

For perspective, Figure 9 plots the incidence of leukemia at Hiroshima and Nagasaki against radiation (90% confidence limits).[7] The incidence of various cancers in the atom-bomb survivors is among the most significant and widely used data on current risk estimates of damage to health. Despite its wide uncertainty (indicated by the spread in the 90% confidence limits and in the uncertainties in the dose actually received by survivors), the data are roughly compatible with linearity between dose and effect. Use of these data for risk estimation of the health damage from the nuclear fuel cycle necessitates extrapolation of damage induced by high doses given at high dose rates down to extremely low doses of radiation given at low rates. Such doses usually comprise a tiny fraction of a percent of natural background radiation; it is impossible to discern any damage to health directly from this natural background. In contrast, the addition of sulfates or related material from fossil fuel combustion, especially in the United States east of the Mississippi, adds to an ambient concentration of sulfates already very close to the level at which clinical damage has been seen. There is thus less uncertainty in this regard in the application of a sulfate-damage function than in the application of the frequently very precisely calculated risk made from radiation.

CRITICISMS BY OTHERS OF THE MULTI-CITY EPIDEMIOLOGICAL STUDIES

Despite the overwhelming evidence linking air pollution and ill-health, many uncertainties still attend the individual studies. Some have argued that certain air pollutants are not bad for health. Special-interest groups have focused on the weaknesses of one or more of these studies. Thus critics analyzing regression studies of city (SMSA) mortality rates, typically of the classic studies of Lave and Seskin, have discussed most of the formal statistical difficulties attending any observational study. The most important criticism is that an observational study only shows association, not causation. The case for the quantitative relation between air pollution and mortality rests on broader studies than observational studies. Eight types of studies connect air pollution and mortality:

1. Multi-city regression studies[10-12] (Lave and Seskin, Hickey et al., Schwing and McDonald);
2. Detailed studies of mortality in a particular city[13-15] (Winkelstein et al. in Buffalo, Zeidberg et al. in Nashville, and Gregor in Pittsburgh);
3. Studies of daily mortality and the association with daily pollution levels in single cities[10,16,17] (Lave and Seskin, Schimmel et al., Buechley);
4. Studies of disastrous air-pollution episodes and consequent increased mortality[18,19] (London air pollution episodes);
5. Animal studies showing damage from air pollution exposure[8,20,21] (Amdur, Frank, and others);

6. Morbidity studies -- in general these have dealt with individuals rather than aggregates[22,23] (Ferris et al., Douglas);
7. Occupational studies (see NIOSH criteria documents on sulfur dioxide and sulfuric acid);
8. Experiments with human subjects[24-26] (Amdur, Frank).

Each type of study has its limitations. However, if one selects a particular limitation, one can find other studies that do not have this limitation.

Table 4 lists some issues that critics can raise. These include presence or absence of randomization, population variables, socio-economic variables, controls for smoking, confounding by "regional effects," and direct applicability to the general population. One row in the table is devoted to each problem, and a column to each method. The code P in a row and column means that the problem specified by this row is not dealt with satisfactorily directly or indirectly by the method specified in this column.

In human epidemiological studies, while experimental randomization (i.e., deciding what pollution conditions an individual will be exposed to without regard for his personal preference but solely on the basis of an arbitrary mechanism, e.g., dice throwing) is necessary for valid causal inference, such randomization is virtually impossible in large-scale, long-term studies because one cannot dictate an individual's living conditions vis-à-vis pollution so arbitrarily. Animal experiments and short-term human studies, however, can be randomized. Such analyses, in fact, show that exposure to specific pollutants damages health in animals and man.

The next three rows in Table 4 exemplify adjustments which, if made in the analysis, might clarify the relationship between air pollution and ill-health: adjustments for the racial and age-sex structure of the population, possible socio-economic variables, and smoking. Since these particular variables are reasonably constant from day to day, the daily studies and studies of air-pollution episodes do not have these as serious limitations, hence have adequately dealt with these problems. These factors are irrelevant in animal studies and in experiments on human beings because appropriate randomization eliminates these issues directly.

The "regional effects" variables only affect the following studies: multi-city, some morbidity, and some occupational. The single-city, daily mortality, and episode studies deal with this problem by being region-specific. The problem is irrelevant in experimental studies on animals and man.

Only the occupational and experimental studies have problems with their direct applicability to the general population.

In sum, there are two or more study methods that address each problem. Workers who have applied the methods in the studies cited above overwhelmingly agree in finding health damage. That human beings are, indeed damaged is dramatically shown by air-pollution episodes, by the association between daily pollution levels and mortality, the multi-city regression studies, and experiments on human volunteers. This consistent mass of evidence impels the assertion that air pollution injures health.

Criticisms of the Lave and Seskin work have raised two questions about the sulfate data. First, that the "1960" data set includes data from earlier years (back to 1957) to fill in missing data for many SMSA's. This is not a problem. Were one studying the relation between daily air pollution peaks and mortality on a given day, substitution of a different day's pollution data would be unacceptable. Lave, however, examines differences in long-term (annual) mortality rates among cities. The effect of pollution on the annual mortality rate of a city depends on the long-term effect of pollution over several years on the general health of the population. Lave's pollution data are an index rather than a specific measure of pollution levels during the year of death.

The second criticism of sulfate data is that bi-weekly and quarterly data are mixed, causing a potential bias in the use of minimum and maximum numbers. This is a valid criticism of Lave and Seskin, but not of the health-damage functions derived by the Biomedical and Environmental Assessment Division (BEAD) at Brookhaven. As documented,[27] the BEAD health-damage function is based on annual average sulfate pollution levels -- an index unaffected by use of bi-weekly or quarterly data. In the BEAD health-damage function, only average sulfate levels are included because our analysis lead us to believe that this was a better base than the minimum or maximum values reported by Lave. Thus, many of the caveats of the critics of Lave and Seskin have already been incorporated in the BEAD analysis.

BEAD PROBABILISTIC ANALYSIS

To mass all uncertainties underlying the quantitative relationship between the operation of a coal-fired power plant and damage to health, Morgan and Morris made a comprehensive probabilistic analysis.[28] Sulfur air pollution transport, dispersion, and impact were modeled from a 1,000-MWe coal power plant to the population within an 80 km radius. A gaussian plume dispersion model with linear sulfur chemistry was used with a linear health-damage function. Important features of the model involving uncertainty were: fraction sulfur emitted as SO_4, SO_2 loss rate, SO_4 loss rate, SO_2 to SO_4 conversion rate, health-damage function, the meteorological model. The uncertainty in each of these variables was characterized by a probability density function based on best available scientific judgment. This provides not only the "estimate" but the estimate of a probability that a variable's actual value may be given in amounts higher or lower than the "estimate." Essentially, these distributions are a description of the odds a knowledgeable scientist might calculate if asked to bet on the outcome of a series of definite experiments which would, at a future time, determine the true value of the variables. These probability density functions (pdfs) were then combined in a simulation analysis to produce estimates of population exposure, premature deaths (Figure 10), and person-years lost.

What are the advantages of characterizing uncertainty as pdfs based on best scientific judgment over the use of classical statistical methods on available data? Several:

1. In many instances, the best data comes from studies which have (often unavoidably) serious flaws, e.g., design problems, unaccounted for variables,

etc. Classical statistical methods cannot clearly interpret results from such studies.

2. Available data are often based on a subset of the total possible universe that may not be representative, e.g., epidemiological studies on certain population subgroups, airborne air chemistry studies on days when the plane can fly, etc.

3. The need to combine results from various kinds of studies, e.g., taking laboratory studies as well as those conducted in the "real world" into account. Extrapolation from laboratory results demands more than classical statistics can provide.

What important uncertainties were not quantified? The model itself and the form of the parameters were assumed to be known. Both may be quite different from that assumed. This was not explicitly dealt with and adds uncertainty beyond that characterized. For example, a linear damage function is assumed and no uncertainty due to possible alternative damage function shape is considered.

THE DEFINITION OF THE HEALTH-DAMAGE FUNCTION

The health-damage function used links annual average sulfate exposure with increased annual mortality rate. It does not represent the acute effects of episodes but the long-term impact on the population of a continuing environmental exposure. Although the sequence of events leading to this impact on the population is unknown, long-term exposure to air pollution, particularly in childhood, presumably increases susceptibility to respiratory infection. A history of repeated respiratory infection, possibly coupled with continued air pollution exposure, increases the prevalence of chronic respiratory disease. This leads to more deaths from a broad range of cardio-pulmonary diseases. Thus increase in air-pollution exposure degrades a population's health; this is eventually reflected in mortality rate. Deaths attributable to an air-pollution exposure in a given year do not necessarily occur that same year, but are distributed over the lifetime of the exposed population. We cannot yet estimate how these deaths are distributed in time. Mortality estimates not only represent premature deaths, but years of decreased respiratory function, perhaps disability before death. Since not all induced respiratory diseases may result in premature death, the annual incidence of new-disease cases is undoubtedly higher than the annual number of deaths. Since the health-damage function is based on annual mortality rates, each death attributed to air pollution represents at least one year of life lost. Reasonable estimates of the age distribution of the deaths leads to the conclusion that 5 to 15 years lost per attributed death are likely.

Under steady-state conditions, the deaths occurring over future years attributable to pollution exposure this year equals the number of deaths occurring this year, due to the summated pollution exposure of all previous years. Based partly on this, a linear health-damage function was drawn from cross-sectional studies as a simplified way to estimate effects of alternative energy strategies. By this simplified linear damage function, incremental sulfate exposure this year inevitably increases health damage and premature deaths. The incremental health damage is proportional to incremental sulfate exposure and is independent of the total sulfate exposure under the linear assumption. These estimated premature deaths may be compared with total deaths annually in the exposed population; this imparts perspective to the estimates. This fraction might be taken as a rough estimate of the eventual contribution to mortality of a continuing air-pollution exposure of that level; it is not the fraction of total deaths attributable to this level of exposure occurring in the same year.

It seems doubtful that the damage function is truly linear with no threshold over the entire range of exposure. More likely, at low absolute levels of exposure, the health impact of a unit increase in sulfate is reduced. There may be a threshold below which there is no detectable health damage. The data on which the dose-response function is based are from urban areas with generally high background sulfate levels but, significantly, the linear function is consistent with data from urban and rural areas with high- and low-pollution levels. Use of a linear function to estimate effects of small changes in sulfate levels in areas with high background levels seems reasonable. Estimates of the effects of sustantial changes in background levels, or of small changes in areas with low initial background levels, increase the uncertainty in estimation of damage.

The health damage function described by Morgan et al.[28] ranges from 0 to

12 deaths per 10^5 per $\mu g/m^3$ sulfate, with a median value of 3.7 (95% confidence interval 0-11.5). These estimates were derived by a subjective analysis of data principally from correlation studies of the type conducted by Lave and Seskin. These studies are subject to methodological and data problems discussed in detail elsewhere. Standing alone, these studies are inadequate to ascribe the observed effect to sulfate air pollution. In concert with toxicological and epidemiological studies, however, they provide a useful means of estimating the magnitude of the damage.

BEAD's OWN ANALYSIS OF AIR POLLUTION AND ILL-HEALTH

Now at the same time as our re-analysis of data available in the literature on the relationship between air pollution and ill-health, we have developed our own data base for this purpose. This includes the total mortality records in the United States for the years 1969, 1970, and 1971 from the National Center for Health Statistics. There are roughly 2 million deaths a year in this file; one is analyzing a total of 6 million deaths for the entire 3,100 counties of the United States, in contrast to the much smaller number of deaths in the standard metropolitan statistical areas (SMSA's) in the Lave and Seskin study. Moreover -- again in contrast to the SMSA studies -- our data include the entire urban and rural portions of the United States. We have also used the 1970 Census Data as a source of some socio-economic variables, especially income. By including three years' total U.S. mortality, and studying people exposed to a wider range of air pollution, one can be that much more confident of the significance of the effects observed.

Our analysis, using these data on the relationship between air pollution and health effects, has proceeded in three stages. In stage 1 all 3,100 counties in the United States were aggregated into 192 groups based on 17 levels of income of the 1970 census and 21 levels of pollution expressed as emissions per square mile. Income variables were represented by race-specific median family income data from the 1970 census. Ideally, in relating air pollution to health effects, one would like to know the actual dose to which the population was exposed. Unfortunately, the dose of air pollution to the population is not available; Lave and Seskin and other studies have used air quality data (concentration of pollutant/m^3) as surrogate for dose. The incompleteness of the air quality data reduces their usefulness initially for a nationwide study of health damage of air pollution. Thus, at this stage in our analysis, we have used estimated emissions as surrogate for dose. This variable varies by five orders of magnitude over urban and rural regions in the United States. Thus, the "pollution" variable was represented by the decimal logarithm of the calculated sulfur emissions (SO_x) in tons per square mile for 1970. This logarithmic transformation of emission has a more normal distribution than the raw estimates and was therefore preferred. The mortality variable was represented by age-, race-, sex-, and cause-specific mortality rates for 1969-1971. The multivariable statistical techniques (multiple regression and path analysis) used provided distinct estimates of the relationship of income level and pollution to mortality. The effects of pollution and income were observed by age cohorts because it was then possible to compute age-, race-, and sex-specific relationships that address the issue of cost in terms of reduction of life span -- not simply total attributable deaths.[29]

A striking feature of the analysis of the relationship of family income to mortality[30] is seen in Figure 11. The average family income in the 3,100 counties for non-whites is less than the average given for whites. This is why we have concentrated in deriving our damage function from data on whites only. One cannot include the non-whites: the impact of income on non-white mortality is just overwhelming. In the 0-4, 5-14, as well as 45-54 age groups there is a striking effect of family income on mortality rate with a notable sex difference in all age groups applicable to non-whites and whites. In the 65-84 age group, as one might expect, the effect of income has leveled off, although the differences in rates between males and females are still apparent.

Figure 12 shows the relationship of income and pollution to mortality for white males and females at all ages. The X-axis contains midpoints of five-year age cohorts. The beta coefficient values for income and pollution as predictors of mortality are graphed on the Y-axis. The beta coefficients -- a standardized regression coefficient in a multiple regression equation -- quantify the strength of the relation between mortality at each age and the indicated variable (income or pollution). A positive coefficient indicates that an increase in the variable goes with increased mortality at that age, while a negative coefficient indicates that increase in the variable goes with a decrease in mortality at that age. Values near 1 or -1 typically represent high association (the beta coefficient may exceed 1 in absolute value, so that beta coefficients should not be interpreted as correlation coefficients). The cross-hatched area indicates where the coefficient is not statistically different from zero.[31]

Income generally tends to be negatively associated with mortality with increasing age. This is particularly evident for white males, in whom income maintains -- and in fact increases -- protection against mortality with advancing age. Pollution for both sexes becomes more strongly associated with mortality as age increases. The only exception is the 0-4 cohort where a positive association with pollution is suspected. This finding suggests that pollution may be especially damaging to infants, as measured by their mortality, and deaths of the very young. Further research is needed.

Comparison of our calculated excess deaths with other research shows striking similarities.[29] Winkelstein et al.[13] attributed approximately 14 deaths/100,000/μg TSP/m^3. Lave and Seskin[10] calculated an order of magnitude lower estimates of 0.9 deaths/100,000/μg TSP/m^3 for white males 55-74. Since both estimates are based on a measure of TSP rather than SO_4, certain modifications of our data were necessary for comparison. The issue of pollution equivalence was a difficult problem. Recent studies have estimated that the SO_4 part of TSP varies by a wide range.[32,33] By assuming 25 to 80 percent to be a reasonable conversion range, we can convert our excess death estimates to figures comparable with the Winkelstein and Lave and Seskin numbers. By a completely different approach, Morgan et al.[28] also calculated excess deaths, however, since their estimates are based on an SO_4 measure of pollution, no modification beyond differentiating between the relative proportion of SO_2 and SO_4 in SO_x was necessary. A modified version of the Finch and Morris[27] comparison of excess deaths is found in Table 5.

Since emissions are not an ideal criterion of air quality in the second stage of our analysis, we have used the air-quality data available in 1970 from the 248 EPA air-quality measurements for three pollutant species: SO_2, SO_4, and total suspended particulates (TSP). As we have already noted, these 248 measuring stations fall far short of covering all 3,100 counties in the United States -- the county was the unit of analysis in the first stage of our analysis; and while major urban areas are monitored by one or more measuring stations, rural areas are sketchily monitored. In an attempt to surmount this problem, i.e., that only 221 counties had one or more EPA air-quality measuring stations in 1970, we have assumed that each EPA measuring station provides good estimates of the pollution level for all those counties which have similar emission and socio-economic characteristics. For this purpose we have used the original 192 groups in the first stage of our analysis to aggregate our data to give a population size large enough to calculate statistically significant mortality rates.[34]

Of these original 192 groups, 92 county groups contained at least one EPA measuring station. In 44 of these 92 county groups, 50% or more of the population reside in counties containing one or more measuring station. Unfortunately, these 44 groups are more representative of polluted urban areas in the U.S. and caution must be exercised when extrapolating these results to the more sparsely represented rural populations. Nevertheless, as will be seen from Figures 13-15, there is, in general, good agreement between the age-specific damage function derived from the emission-mortality analysis from stage 1 of our analysis and that obtained by using EPA measuring stations and grouping emissions in counties without measuring stations with these, stage 2 of our analysis. The data agree reasonably with those derived by Lave and Seskin and Morgan et al. for SO_4 and with Winkelstein and Lave and Seskin for TSP. The difference in the damage function for SO_4 exposure derived from stage I and stage II in age groups over 60 may be related to differences in the size of the populations included in each stage, and the underrepresentation of the rural population in stage II.

For perspective, it is worth noting that the National Academy of Sciences, in its report on saccharin, estimated the risk associated with the equivalent of the consumption of one can of diet soda per day. Depending on different mathematical models used, the estimate of risk varied by five orders of magnitude.

LOW-LEVEL RADIATION EFFECTS

Except for accidental cases of acute high-level exposure, worries about nuclear facilities center on possible damage by airborne or liquid effluents and resultant low radiation doses to people nearby. Note almost all delayed cancers from power-plant accidents in WASH-1400 are calculated to be also due to low radiation doses (<1 rad).

What -- if any -- are levels "safe," "acceptable," and understandable to the public? But neither term can be quantitatively defined. In practice both imply a definite but extremely small probability of risk.

There are now three ways of determining what dose-levels may be considered tolerable. One determines at what dose level the risk of detectable somatic or genetic radiation damage becomes low enough to be vanishingly small in comparison to the summated environmental insults to somatic and gonadal cells due to "normal" causes encountered in a person's lifetime, including background radiation, i.e., to nuclear radiation that forms part of our natural environment, e.g., from potassium and cosmic rays, and determines below what level any additional radiation exposure would result in an insignificant increment in dosage. A cut-off or de minimus level of risk (and therefore of dose) has also been suggested for general application to the general population. Deaths of 1×10^{6} to 1×10^{-5} per individual per year have been proposed.

The difficulties in defining the effects of very low levels -- a few millirem per year or less -- on the general population from the nuclear-fuel cycle are two-fold. For one, no statistically valid experimental work is at hand because of the virtually insurmountable difficulties imposed by the necessarily large-scale, long-term nature of such experiments, e.g., a population of P million will have about P million live born children per generation with ~3.5% of them obviously genetically defective. Thus in several, say 3 generations there will be 10^5 P such defectives in the control population. The population exposed at a few, say 3, mrem y^{-1} will have had an excess exposure of 0.10 rem by the mean age of child-bearing or conception (~30 y). With an induction rate of genetic defects by radiation of 80×10^{-6} rem^{-1}, a population of P million will in 3 generations have had an excess of induced defectives of $3P \times 10^6 \times 80 \times 10^{-6} \times 0.1 = 24$ P extra defectives. For detection of this excess at 2 standard errors $24\ P = 2 \sqrt{(10^5 + 24)\ P}$. Hence P = 700 million. Conditions on a very large, genetically extremely homogeneous population would have to be rigorously controlled and over several generations to detect significant genetic changes. Even epidemiological studies on comparable populations living exposed to different natural radiation levels have proved inconclusive. It also would be imperative to establish a unique cause-and-effect relationship--even were correlations between morbidity and radiation exposure apparent--in order to rule out other causes, such as economic conditions, nutritional factors, or improved diagnostic facilities due to better medical services.

Another problem arises from the fact that, although it is easy to observe deaths and malignancies such as observable at dose levels above 50-100 rad, it is more difficult to observe minor incidences of illness in a much larger population, such as might result from low-level exposures, and to distinguish them unambiguously from similar illnesses that could be caused by a host of other causes.

Due to these difficulties, a conservative approach is to take the dose-effect values obtained at high-dose levels and to extrapolate them down to low-dose levels. What is usually assumed is a linear relationship between dose and effect, independent of dose rate, an assumption that appears to be amply justified for acute high dose levels.

Several recent reports (Bross;[35] Mancuso, Stewart and Kneale;[36] and Najarian[37]) have been interpreted by some people to indicate that the commonly employed risk estimates, which are based on the UNSCEAR and BEIR Committee Reports, underestimate the risk of radiation at all levels. They especially emphasize that the linear theory (that the risk per unit dose as derived from available data at high levels of radiation dose holds all the way down to zero exposure dose) is not sufficiently conservative in estimating risk at low doses but rather underestimates it.

Bross believes he has identified subgroups in the population which are espcially sensitive to radiation damage. His belief derives from his analysis of the Tri-State Leukemia Survey, wherein he studied an association between some "indicators of susceptibility" (viral infections, bacterial infections, and allergy) shown by the leukemic child from birth until diagnosis of leukemia. He concluded "the apparently harmful effects of antenatal irradiation are greatly increased in certain susceptible subgroups of children possessing the indicators associated with a slightly higher intrinsic risk of leukemia." However, reanalysis[38] of his findings shows that children with leukemia are simply more prone to viral and bacterial infections and allergies before the clinical onset of the disease, i.e., these indicators characterize the disease itself and do not relate to the child's inherent susceptibility to leukemia. The incidence of these diseases as part of the pre-leukemia phase of leukemia in children is well known in clinical hematology. Analysis of

Bross' data shows that the incidence of these indicator diseases before the clinical onset of leukemia is the same in children who had received no irradiation in utero as in those who had.[38] The hypothesis of Bross, that there is a susceptible portion of the population at higher risk of leukemia, has also been challenged on the grounds that Bross' methods yield no way to identify susceptible individuals ahead of time and so no way to test his thesis.[39]

More recently, Bross has suggested that the relatively small radiation exposures from diagnostic X-rays in adults significantly increases the risk of leukemia.[40] It appears that Bross assumes, in coming to this conclusion, that in the absence of diagnostic X-rays, the incidence of heart disease and leukemia is zero.[40,41] Were this not the case the fact that the "dose-response" curves of adults exposed to diagnostic X-rays are flat below 10 rad exposure would suggest a threshold. Indeed, a more conventional relative risk analysis[42] found little or no increase in risk of leukemia from a small number of diagnostic X-rays. Bross also assumes here that relative risks are fixed and that the percentage of the population affected varies with dose, i.e., the basic response variable is the proportion of the irradiated population affected by radiation. Conventional relative risk analyses assume that everyone is affected and that the relative risks vary with dose. The improvement made by Bross et al.'s approach is unclear. The position taken here by Bross appears to be at odds with his earlier paper, in which he postulated the existence of a sensitive subgroup of fixed size whose relative risk of leukemia increased rapidly with increasing X-ray dose.

Finally, one should note that the leukemia risk (or "percent affected") does increase dramatically in males (females appear to be unaffected) after large numbers of diagnostic X-rays. However, the cause-effect relationship is uncertain in that large numbers of diagnostic X-rays — ~ 40 or more within 10 years -- implies the presence of a disease state perhaps deriving from heart disease or a preleukemic sensitivity to infections.

Mancuso, Stewart, and Kneale[36] have reported preliminary findings on the work and survival experience of 24,939 male workers with 3,520 certified deaths and of an unspecified number of female workers with 412 certified deaths at the Hanford Works, Richland, Washington between 1943 and 1971. The preliminary report, largely limited to analysis of data on the 3,520 male deaths for which death certificates were available, claim to demonstrate a radiation-induced excess of cancers, greater than linear models would indicate. Their analysis has been widely criticized. Their report does not state the actual individual doses received by Hanford workers who died of cancer, only mean cumulative radiation doses. Besides, their study did not take into account the calendar year in which the cancer began and made no correction for the fact that the incidence of the cancers they were observing in the Hanford workers also increased during the period of the study in the population at large. Thus, Table 11 in their publication, showing an increase in cancer with increasing dose accumulated over increasing time, fails to take into account that even in the absence of the increasing dose of radiation, there is a similar increase in cancers they were finding in the U.S. as a whole when plotted against increasing time. Other analyses of the same data published by Marks et al.[43] and by Hutchinson et al.[44] point to the possibility of an association with the work experience for two cancer types: cancer of the pancreas and multiple myeloma (multiple myeloma in whites is increasing in the U.S. for no known reasons). There is no reported radiation relationship for lymphatic or haemopoietic cancers other than myeloma, i.e., no excess of leukemias (which previous experience suggests should have been most observable where radiation is a factor).

Since the specified radiation doses were very small, perhaps on the order of a few rads, the cancer-doubling estimates found in the Mancuso, Stewart, and Kneale paper have been strongly disputed. If the postulated small dose actually caused a doubling of the spontaneous rate of cancers, then background radiation would produce more than the numbers of cancer observed in the population. It therefore appears that if these doubling doses are correct, something other than radiation was the cause of the observed cancers. In the light of these criticisms, Mancuso et al. now appear to have modified their estimates of doubling doses and are quoting doubling doses of 2-150 rem.

Najarian and Colton[37] estimated that since the Portsmouth Naval Shipyard (PNS) in New England began to service nuclear-powered ships in 1959, 20,000 people were employed there, of whom about 20% were exposed to radiation. From a search of death certificates 1959-77, 1,450 former PNS employees who had died below age 80 were identified in New Hampshire, Maine, and Massachusetts. To ascertain whether these ex-employees were radiation-exposed workers, attempts were made to contact near relatives by telephone. This was successful in 525 cases and it was established that 146 were probably exposed to radiation during their working life.

The authors show that, compared with mortality in U.S. white males for 1973, the observed numbers of cancers and leukemias were considerably greater than those expected: for example, 56 cancer deaths were found in death certificates of 146 ex-workers exposed to radiation; only 34.5 were expected.

In non-exposed workers there were 88 cancers; 79.7 were expected. For leukemias there were 6 in the former radiation workers; only 1.1 were expected.

Najarian and Colton listed some inadequacies in their survey. It was an analysis of deaths only; no information was available on the total population at risk. There could be a bias in the information supplied by relatives. They had no information on how long workers worked at the shipyard, how long nuclear workers were exposed to radiation, and the amounts of radiation they received. Consideration was not given to other toxic agents, such as asbestos, smoking, industrial solvents which could have acted alone or synergistically with radiation to cause the apparent excess deaths from cancer and leukemia.

There are other inadequacies in this survey. To exclude some of the effects of other carcinogens, one must show that cancer frequencies increase with increasing radiation exposure, but knowledge of the lifetime accumulated doses of the former employees was not available. More importantly, if the radiation work at PNS began only in 1959, it is unlikely that changes in overall cancer frequency induced by radiation would appear before at least 10 years after exposure, or after 5 years for leukemia, these being roughly minimum latent periods for cancer induction. The data given in Najarian and Colton can be divided into deaths during the periods from 1959-69, when radiation effects would not be expected to appear, and 1970-77, when effects might be expected. In 585 death certificates of persons who died between 1959-69, 24.6% had cancer listed as the cause of death. Considering the 33 radiation-exposed workers who died during this period, 13 or 39.4% of the deaths were recorded as due to cancer. In 865 death certificates 1970-77, 25.7% had cancer as the cause of death; hence there was no significant difference between the percentage of cancer deaths between the two periods for all workers. For the 113 radiation-exposed workers, 43 or 38.1% of deaths in the later period where due to cancer -- no more than in the earlier period (39.4%).

				RADIATION EXPOSED		
	ALL DEATHS	CANCER DEATHS	% CANCER DEATHS	ALL DEATHS	CANCER DEATHS	% CANCER DEATHS
1959-69	585	144	24.6	33	13	39.4
1970-77	865	222	25.7	113	43	38.1
	1,450	366				

The absence of any apparent latent period effect casts doubt on conclusions about the contribution of radiation to the curiously high numbers of cancer deaths among the radiation workers.[45]

In the meanwhile, NIOSH made available to Drs. Najarian and Colton radiation exposure data supplied by the U.S. Navy. On February 2, 1979, at a symposium sponsored by the Johns Hopkins School of Public Health, Baltimore, Maryland, Drs. Najarian and Colton introduced these radiation exposures into their PNS Study. At this time, they announced that in contrast with the original Lancet data, where 6 leukemia deaths were observed instead of 1.1 expected, it was found that two of the cases of leukemia had no history of radiation exposure. One had less than 0.1 rem, which is what one receives after one year's natural background. One received 15 rem, one 5 rem, and one "not numbered" -- probably less than 5 rem. The number of leukemias is now 3 instead of 1.1 expected. For all cancers the new data are:

	CANCERS			
EXPOSURE	NUMBER	OBSERVED	EXPECTED	RATIO
Less than 0.1 rem	64	17	13.5	1.26
From 0.1 to 0.99	50	16	10.5	1.53
Greater than 1	49	19	10.2	1.58
No exposure	358	92	7.49	1.24

Chi-square test shows no significant difference in the ratio among the exposed levels at p = 0.10. Cochran's chi-square test for a linear regression, which considers that the ratios increase in the expected direction shows no statistical significance at p = 0.05 but is significant at p = 0.10.

Early verification of these data is urgent.

A tentative list* of chemical and physical agents probably present at the Portsmouth Naval Shipyard during the past 25 years includes:

Acetone
Alcohols
Amines
Ammonia
Ammonium Hydroxide
Amyl Acetates
Antimony Oxide
Asbestos
Benzene
1, 3, Butadiene
Cadmium
Carbon Monoxide
Carbon Tetrachloride

Chlorinated Diphenyls
Chlorinated Napthalenes
Chromic Acid
Chromium
Coal Tar Volatiles
Epichlorohydrin
Ethyl Acetate
Ethylenediamine
Fibrous Glass
Fluorides
Fungicides
Hydrogen Chloride
Hydrogen Cyanide

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* Personal communication: R.D. Zumwalde

Infrared light

Lead

Methyl Ethyl Ketone

Methyl Isobutyl Ketone

Mineral Wool

Nickel

Phenols

Polyvinyl Chloride

Radiofrequency (Microwave)

Silica (Quartz)

Sodium Hydroxide

Titanium Oxide

Toluene

Trichloroethylene

Tri Sodium Phosphate

Turpentine

Ultra Violet Light

Wood Preservatives

Xylene

Zinc Chromate

Comparison with Table 6, taken from Table 25: Common Occupational Carcinogens from Health and Work in America: A Chart Book, American Public Health Association, Washington, D.C., 1975, underscores the difficulty in assessing the effects of low levels of radiation in this worker population.

One must await the results of the complete survey being carried out by the Center for Disease Control and National Institute for Occupational Safety and Health before any conclusions can be drawn between exposure to radiation at PNS and risk of cancer.

Thus, although these claims of higher risks from the levels described by Bross, Mancuso, Stewart, Kneale, and Najarian have become the subject of considerable public debate, examination to date of their work does not support these claims.

BEAD'S APPROACH TO ASSESSMENT IN THE FACE OF UNCERTAINTIES

BEAD at Brookhaven is assessing the health and environmental costs of energy production and use. BEAD's methodology is straightforward. One starts with compilation of pollutants from the energy system, then the various pathways to man are traced. This task entails definition of transport mechanisms, including chemical and biological conversions and varous transfers through the biosphere to man. A quantitative evaluation is then made of effects. This evaluation relies on information from epidemiological data, field and laboratory studies, and biomedical research designed to elucidate molecular and cellular mechanisms underlying responses to pollutants. Once given the magnitude of energy flows through the system and the size of the populations exposed, total health damage can be estimated. The framework of the evaluation is provided by the energy-system models of the National Center for Analysis of Energy Systems, of which BEAD is a part; this provides the resources (including the required energy-flow projections) and the means of integrating the results with a national energy policy.

Most energy models are based on aggregated national data or on large regions, such as census regions. Health and environmental effects, however, are highly location-dependent. The need for a high degree of geographical disaggregation of emission data led us to develop a county-level energy and emissions data base.[46] We maintain a file on sites of planned future energy facilities. For assessment, the Regional Studies Division at the Center has developed methods for siting future energy facilities associated with long-term energy projections. Data on emissions from these sites are then fed into large-scale meteorological-air chemistry transport models[47] to estimate likely ambient air quality.

Risk assessment aims at improving the base for decision-making for policy makers and public. The information upon which to base risk assessment is often scanty. Even so, assembling available data, organizing them so as to clarify choices among energy options, and gauging the uncertainties is a useful aid to decision-making. Since decisions mean choices among different technologies, standardized comparisons are essential to avoid poor choices. For technologies producing the same form of energy, e.g., electricity, a standardized unit of production or production rate can be used, e.g., a 1,000-MWe power plant-year. When comparisons must extend across technologies producing different energy forms, e.g. coal-electric vs. coal-gasification vs. coal-liquefaction, the proper comparison is not always obvious. Indeed, there may not be a totally satisfactory basis. Streams of electricity, gas, and oil with the same energy content are not really equal: they are used by the consumer for different purposes and with different efficiencies. As discussed in the Introduction, this difficulty can be largely overcome by examining different technological choices within the entire energy system meeting all demands (Figure 2). Risk assessment must quantitatively assign risk to each component of the energy system; valid comparisons can be made only between entire fuel cycles or between entire alternative energy systems. While we are not yet able to analyze completely environmental and health impacts with quantitative data for the entire energy system, current economic- and technology-oriented models use this integrated framework.[48]

The main difficulty in risk assessment lies in comparison of qualitatively different risks. There may be trade-offs among air, water, and land impacts; between short-term and long-term risks; or between extraordinary and routine risks. In comparing coal and nuclear fuel cycles, for example the high probability of air pollution impacts from coal (although uncertain in magnitude) must be compared with the low probability of catastrophic events from nuclear energy. Quantitative analysis alone cannot tell how large a certain, routine impact one is willing to accept to avoid an unlikely catastrophe. Risk assessment would help separate scientific issues from value issues in these very thorny technological problems, thus helping the public and political decision-makers clarify key value decisions.

Until recently, we concentrated on comparisons of the main sources of electric power. Tables 7 and 8 summarize our preliminary estimates of health effects on a unit-plant basis,[49] assuming currently mandated environmental controls. Table 7 shows a summary of the nuclear cycle effects on a unit-plant basis. Table 8 shows our current estimates of the health effects of the coal fuel cycle on a unit-plant basis. Most of our attention has been directed to quantifying coal-mining accidents and occupational disease, coal transport accidents, and air pollution from coal combustion.

Detailed Approach to Assessment

Some of the results of our assessments are incorporated into a coordinated effort of several groups within the national laboratories in support of a U.S. Department of Energy program called the National Coal Utilization Assessment (NCUA).[50] The complete project is wide-ranging. It may be useful to the Ohio River Basin Energy Study (ORBS) to examine what -- from our point of view -- is the main theme of the exercise, since the course of ORBS must be somewhat similar. It consists of six steps:

1. Scenarios which span the range of energy policies to be analyzed for specific future reference years were

designed.

2. Energy technology-economic analysis at the regional level resulting in estimates of energy supply, conversion, and use by fuel, sector, and region was carried out.
3. Specific energy facilities were sited within each region. This was done with models which consider historical trends, resource availability, end use, etc.
4. Environmental transport models were run to convert air-pollutant emissions to ambient concentrations. The result was a geographical distribution of air-quality contributions from energy use in each scenario-reference year.
5. Population exposures were determined by overlaying the pollution distribution produced in step 4 on mapped population distribution. The result was the number of people exposed to various levels of energy-generated pollution. Although this has been applied only to man, the same approach is expected to estimate pollution exposure to crops and various natural ecosystems.
6. Using dose-response functions developed independently, the impact of population exposure was estimated.

Several aspects of this approach are under further study. Some examples: (a) background pollution exposures from non-energy sources are an important consideration that should be projected and analyzed -- attempts have been made to do this but it greatly enlarges the required effort; (b) more opportunity to consider environmental factors and alternative emission controls within the technology-economic modeling of step 2; (c) opportunities to allow changes in the technology-economic modeling based on the impacts calculated in step 6; (d) expansion of the kinds of environmental modeling -- we are now working on water-quality modeling; (e) expansion of receptor categories - mappings of agricultural crops and endangered species are being integrated with pollution level and siting mappings to identify potential impacts; (f) flexibility -- each step is an independent process, products are available for review after each step, and revision of the models in any one step can be easily incorporated.

Simplified Approach to Assessment

The comprehensive approach requires much time, personnel, and coordination. Frequently, quick, first-cut estimates must be made of the effects of various policy options. To meet this need, we have used a unit effects approach. Based on detailed case studies, we have developed impact estimates for typical unit facilities, particularly power plants. If a scenario calls for the equivalent of 50 1,000-MWe coal power plants, we assign the impact to be 50 times the impact of our unit plant. We have used the unit effects approach on national, regional, and multi-regional studies.

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Table 1 MECHANISMS THAT CONVERT SULFUR DIOXIDE TO SULFATES

Mechanism	Overall reaction	Factors on which sulfate formation primarily depends
1. Direct photo-oxidation	$SO_2 \xrightarrow[\text{water}]{\text{light, oxygen}} H_2SO_4$	Sulfur dioxide concentration, sunlight intensity.
2. Indirect photo-oxidation	$SO_2 \xrightarrow[\text{organic oxidants, hydroxyl radical } (OH^{\cdot})]{\text{smog, water, } NO_x} H_2SO_4$	Sulfur dioxide concentration, organic oxi-oxidant concentration, $OH^{\cdot}$; NO_x
3. Air oxidation in liquid droplets	$SO_2 \xrightarrow{\text{liquid water}} H_2SO_3$ $NH_3 + H_2SO_3 \xrightarrow{\text{Oxygen}} NH_4^{+} + SO_4^{=}$	Ammonia concentration
4. Catalyzed oxidation in liquid droplets	$SO_2 \xrightarrow[\text{heavy metal ions}]{\text{oxygen, liquid water}} SO_4^{=}$	Concentration of heavy metal (Fe, Mn) ions
5. Catalyzed oxidation on dry surfaces	$SO_2 \xrightarrow[\text{carbon, water}]{\text{oxygen, particulate}} H_2SO_4$	Carbon particle concentration (surface area)

TABLE 2

INCREASE IN MORTALITY IN THE LONDON FOG OF DECEMBER 1952

CAUSE OF DEATH	SEASONAL NORM (DEATHS PER WEEK)	DEATHS IN WEEK AFTER FOG	EXCESS DEATHS	PERCENTAGE OF TOTAL EXCESS DEATHS
Bronchitis	75	704	629	39
Other lung diseases	98	366	268	17
Coronary artery disease, myocardial degeneration	206	525	319	20
Other diseases	508	889	381	24
Total	887	2484	1597	100

Table 3 – Ranking of sulfates for irritant potency.

Compound	% Increase Resistance/μg SO_4/m^3
H_2SO_4	0.410
$Zn(NH_4)_2(SO_4)_2$[1]	0.135
$Fe_2(SO_4)_3$[2]	0.106
$ZnSO_4$[1]	0.079
$(NH_4)_2SO_4$[1]	0.038
NH_4HSO_4	0.013
$CuSO_4$	0.009
$FeSO_4$[2]	0.003
$MnSO_4$[2]	−0.004

[1] Data of Amdur and Corn (1963).

[2] Data of Amdur and Underhill (1968).

TABLE 4

Enumeration of Strengths and Weakness Study Designs
for
Air Pollution Health Effects

	Multicity	Single City	Daily	Morbidity	Episode	Occupational	Controlled Human	Animal
Randomize	P	P	P	P	P	P	N	N
Population	P	P	N	P	N	P	N	NA
Socio-Economic	P	P	N	P	N	P	N	NA
Smoking	P	P	N	P	N	P	N	NA
Direct Applicability to General Population	N	N	N	N	N	P	P	P
Regional Effects	P	N	N	P	N	P	N	N

P = possible problem area

N = design directly or indirectly controls for this factor

NA = not applicable

TABLE 5

Comparison of Attributable Pollution Death Estimates by Various Researchers

Researchers	Excess Deaths (death x 10^5/ug pollution type)	95% Confidence Limit Minimum	 Maximum
Winkelstein (1967)***			
white males and females			
ages 50-69 ug/TSP/m^3	14.00	10.00	18.00
Bozzo et. al.			
white males and females			
ages 50-69			
ASSUMPTION: 25% SO_4/TSP	2.29	0.91	3.67
80% SO_4/TSP	7.34	2.91	11.76
Lave and Seskin (1977)*			
white males and females			
ages 45-64 ug/TSP/m^3	0.90	0.40	1.40
Lave and Seskin (1977)			
white males 45-64, (ug SO_4/m^3)	4.41	-5.80	14.60
white females 45-64 (ug SO_4/m^3)	8.10	2.20	14.00
Bozzo et. al.			
white males and females			
ages 45-64			
ASSUMPTION: 25% SO_4/TSP	1.68	0.68	2.69
80% SO_4/TSP	5.38	2.16	8.59
Lave and Seskin (1977)			
All ages, white male	4.80	-0.50	10.10
All ages, white female	9.35	4.50	14.20
Morgan et. al. (1977)			
all ages (ug SO_4/m^3)	3.71**	0.00	11.47
Bozzo et. al. (1977)			
all ages (ug SO_4/m^3)	3.56	1.16	5.96

* Personal communication to S. Finch (in Finch and Morris 27)

** Median value

*** As calculated by Finch and Morris from Winkelstein's data

Common cancer inducing agents have been linked to various occupational groups.

Table 6
COMMON OCCUPATIONAL CARCINOGENS

Agent	Organ Affected	Occupation
Wood	Nasal cavity and sinuses	Woodworkers
Leather	Nasal cavity and sinuses; urinary bladder	Leather and shoe workers
Iron oxide	Lung; larynx	Iron ore miners; metal grinders and polishers silver finishers; iron foundry workers
Nickel	Nasal sinuses; lung	Nickel smelters, mixers, and roasters; electrolysis workers
Arsenic	Skin; lung; liver	Miners; smelters; insecticide makers and sprayers; tanners; chemical workers; oil refiners; vintners
Chromium	Nasal cavity and sinuses; lung; larynx	Chromium producers, processors, and users; acetylene and aniline workers; bleachers; glass, pottery, and linoleum workers; battery makers
Asbestos	Lung (pleural and peritoneal mesothelioma)	Miners; millers; textile, insulation, and shipyard workers
Petroleum, petroleum coke, wax, creosote, shale, and mineral oils	Nasal cavity; larynx; lung; skin; scrotum	Contact with lubricating, cooling, paraffin or wax fuel oils or coke; rubber fillers; retort workers; textile weavers; diesel jet testers
Mustard gas	Larynx; lung; trachea; bronchi	Mustard gas workers
Vinyl chloride	Liver; brain	Plastic workers
Bis-chloromethyl ether, chloromethyl methyl ether	Lung	Chemical workers
Isopropyl oil	Nasal cavity	Isopropyl oil producers
Coal soot, coal tar, other products of coal combustion	Lung; larynx; skin; scrotum; urinary bladder	Gashouse workers, stokers, and producers; asphalt, coal tar, and pitch workers; coke oven workers; miners; still cleaners
Benzene	Bone marrow	Explosives, benzene, or rubber cement workers; distillers; dye users; painters; shoemakers
Auramine, benzidine, alpha-Naphthylamine, magenta, 4-Aminodiphenyl, 4-Nitrodiphenyl	Urinary bladder	Dyestuffs manufacturers and users; rubber workers (pressmen, filtermen, laborers); textile dyers; paint manufacturers

SOURCE: National Cancer Institute.

TABLE 7

NUCLEAR FUEL CYCLE EFFECTS SUMMARY

	EFFECTS PER 0.65 GWY(E)	
	DEATHS	DISEASE/INJURY
MINING		
PUBLIC	0.08	0.08
WORKERS		
RADIATION INDUCED CANCER	0.06	0.03
NON-RADIATON INDUCED OCCUPATIONAL DISEASE	0.07	0.14-2.8*
OCCUPATIONAL ACCIDENTS	0.31	11.96
SUBTOTAL	0.52	12.21-14.87
PROCESSING		
PUBLIC	0.002	0.002
WORKERS		
RADIATION INDUCED CANCER	0.034	0.034
OCCUPATIONAL ACCIDENTS	0.004	1.3
SUBTOTAL	0.04	1.34
ELECTRICITY GENERATION		
ROUTINE PUBLIC	0.017	0.017
WORKERS		
RADIATION INDUCED CANCER	0.07	0.07
OCCUPATIONAL ACCIDENTS	0.013	1.13
CATASTROPHIC ACCIDENTS	0.1	---
SUBTOTAL	0.20	1.217

*BASED ON RATIO OF OCCUPATIONAL DISEASE/DEATH IN COAL MINERS. LOWER ESTIMATE IS USED IN TOTAL.

TABLE 7
NUCLEAR FUEL CYCLE EFFECTS SUMMARY (CONTINUED)

WASTE MANAGEMENT		
PUBLIC	5.1×10^{-5}	5.1×10^{-5}
WORKERS	7.45×10^{-4}	7.45×10^{-4}
SUBTOTAL	7.96×10^{-4}	7.96×10^{-4}
TRANSPORT		
ROUTINE PUBLIC	6.1×10^{-4}	6.1×10^{-4}
WORKERS		
RADIATION INDUCED CANCER	8.5×10^{-4}	8.5×10^{-4}
OCCUPATIONAL ACCIDENTS	0.01	0.1
CATASTROPHIC ACCIDENTS		
CANCERS	8.3×10^{-5} TO 7.1×10^{-4}	8.3×10^{-5} TO 7.1×10^{-4}
PROMPT DEATHS	2.1×10^{-7} TO 9.3×10^{-5}	
SUBTOTAL	0.01	0.10
DECOMMISSIONING		
PUBLIC	5.3×10^{-9}	5.3×10^{-9}
WORKERS		
RADIATION INDUCED CANCER	4.2×10^{-3}	4.2×10^{-3}
OCCUPATIONAL ACCIDENTS	8.0×10^{-4}	0.07
SUBTOTAL	5×10^{-3}	0.07
TOTAL	0.77	14.9-17.6

TABLE 8

COAL FUEL CYCLE EFFECTS SUMMARY
(PER 1,000 MW PLANT-YEAR, 65% CAPACITY)

	DEATHS	DISEASE/INJURY
MINING[1]		
PUBLIC	---	---
WORKERS		
ACCIDENTAL INJURY[2]	0.6	42
OCCUPATIONAL DISEASE	0.02-0.4	0.5-1.0
PROCESSING		
PUBLIC		
WORKERS		
ACCIDENTAL INJURY	0.05	2.9
OCCUPATIONAL DISEASE	---	---
TRANSPORT[3]		
PUBLIC AND WORKERS		
ACCIDENTAL INJURY	0.3-1.3	1.2-5.9
ELECTRICITY GENERATION		
PUBLIC		
AIR POLLUTION (50 MI RADIUS)[4]	0.6 (0-3)	NOT ESTIMATED
AIR POLLUTION (TOTAL U.S.)[5]	6 (0-30)	NOT ESTIMATED
WORKERS		
ACCIDENTAL INJURY[6]	0.1 (0.02-0.3)	3.3 (2.7-4.0)
TOTAL	7.7-9.1	

TABLE 8

COAL FUEL CYCLE EFFECTS SUMMARY (CONTINUED)

NOTES:

1. ASSUMES 62% UNDERGROUND, 38% SURFACE MINING (THE RATIO OF APPLACHIAN COAL PRODUCTION, SOURCE U.S. BUREAU OF MINES, MINERAL YEARBOOK 1974, U.S. GOVERNMENT PRINTING OFFICE, 1976, VOL. 1, PP. 367-76).

2. COAL MINERS ACCIDENTAL (NON-FATAL) INJURY (1965-73 MEN)

   UNDERGROUND MINING - 27.6 INJURIES PER 10^6 TONS
   SURFACE MINING - 5.2 INJURIES PER 10^6 TONS

   $$\left[(27.6 \times 0.62) + (5.2 \times 0.38)\right] \times 2.2 \times 10^6 = 42 \text{ INJURIES PER PLANT-YEAR}$$

   FROM MORRIS AND NOVAK (REF. 61, P.13)

3. ASSUMES RAIL TRANSPORT, 300 MILE TRIPS. RANGE IS DUE TO DIFFERENT METHODS OF ESTIMATION.

4. ASSUMES 3 MILLION PEOPLE WITHIN 50 MILE RADIUS, SULFUR OXIDE EMISSION RATE OF 0.12 LBS. So_2 PER 10^6 BTU INPUT (LOW SULFUR COAL COMBINED WITH 90% REMOVAL OF SULFUR IN FLUE GAS. RESULTS ARE APPROXIMATELY LINEAR FOR SO_2 EMISSIONS.

5. ASSUMES TOTAL EFFECT 10X LOCAL EFFECT.

6. ESTIMATES FROM BERTOLETT AND FOX, WITH POISSON 95% CONFIDENCE LIMITS.

LEGENDS TO FIGURES

1. The position of biomedical assessment in overall energy assessment.
2. Reference energy system, 1977, diagramming stages in energy production, distribution and use.
3. Deposition in various portions of the lung and respiratory tract for various size particle populations. Nasal-P stands for the nasopharyngeal compartment, T-Bronchial for the tracheobronchial compartment, and Pulmonary for the alveolar compartment.
4. Correlation between the prevalence of chronic bronchitis and sulfur oxide precipitation (measured by PbO_2 method) for differing smoking rates in the subjects after correction for sex and age.
5. Total number of deaths for 156 winter weeks in Oslo, Norway (1958/9 to 1964/5) plotted against weekly mean concentration of SO_2.
6. Excess acute lower respiratory disease in children plotted against annual average suspended sulfates concentration.
7. Aggravation of heart and lung disease in the elderly plotted against 24-hour suspended sulfates concentration.
8. Excess mortality rate in New York City in 1960 plotted against 24-hour suspended sulfates concentration (includes data on excess mortality in London in 1950's and Oslo in 1960's).
9. Excess leukemia mortality at Hiroshima and Nagasaki plotted against the dose in rem (with 90% confidence limits). Kerma dose was converted to effective absorbed dose. (The line is a least-square fit forced through the origin deleting the two negative Nagasaki points. The slope is 49.90 deaths/10^6 person-rem with a standard deviation of 4.27.)

10. BEAD probability density functions representing the uncertainty in the slope of the assumed linear damage function. The solid curve is the subjective distribution. The dashed curve is the classical estimate (student's _t_ distribution with three degrees of freedom) obtained by treating the results of four regression studies as independent observations of the same slope.

11. Sex-, race-, age-specific mortality rates and family income, 1970.

12. The combined effects of income and pollution by age for white males and females, total mortality.

13. Total deaths associated with SO_4 exposure (deaths/100,000/μg/SO_4/year), U.S. 1969-71 white males. Stage 1: Analysis based on emissions data. Stage 2: Analysis based on air quality and related emissions data. Note the average values given by Lave and Seskin and the results of the probabilistic study from Morgan et al. for both sexes at all ages.

14. Total deaths associated with SO_4 exposure (deaths/100,000/μg/SO_4/year), U.S. 1969-71 females. Stage 1: Analysis based on emissions data. Stage 2: Analysis based on air quality and related emissions data. Note the average values given by Lave and Seskin and the results of the probabilistic study from Morgan et al. for both sexes at all ages.

15. Total deaths associated with total suspended particulates exposure (deaths/100,000/μg/TSP/year), U.S. 1969-1971 white males. Stage 1: Analysis based on emissions data assuming TSP contains 25% SO_4. Stage 2: Analysis based on air quality and related emissions data. For comparison note values given by Winkelstein for age 50-69 and Lave and Seskin for age 45-64.

ENERGY POLICY ASSESSMENT STRESSING BIOMEDICAL EFFECTS

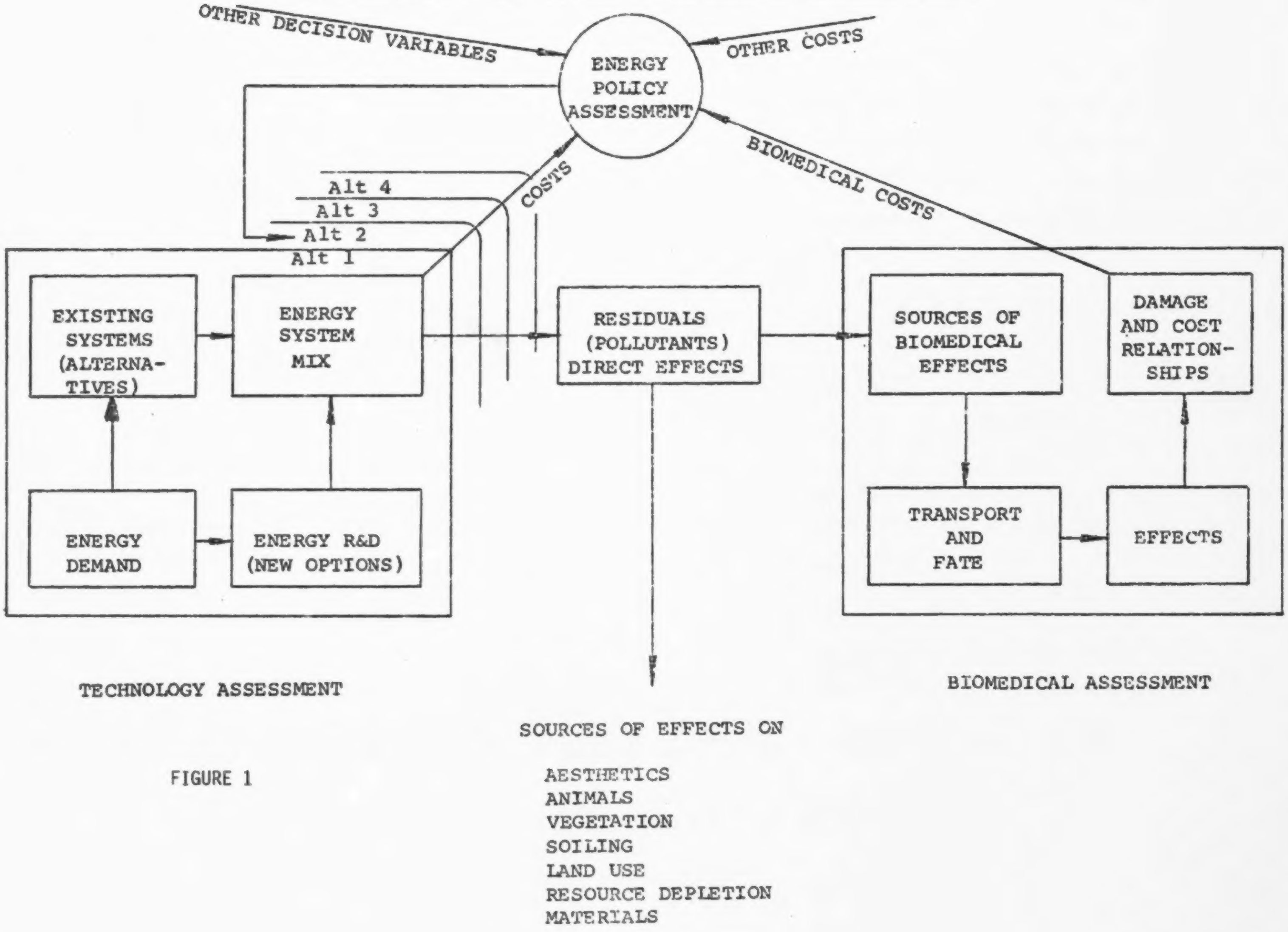
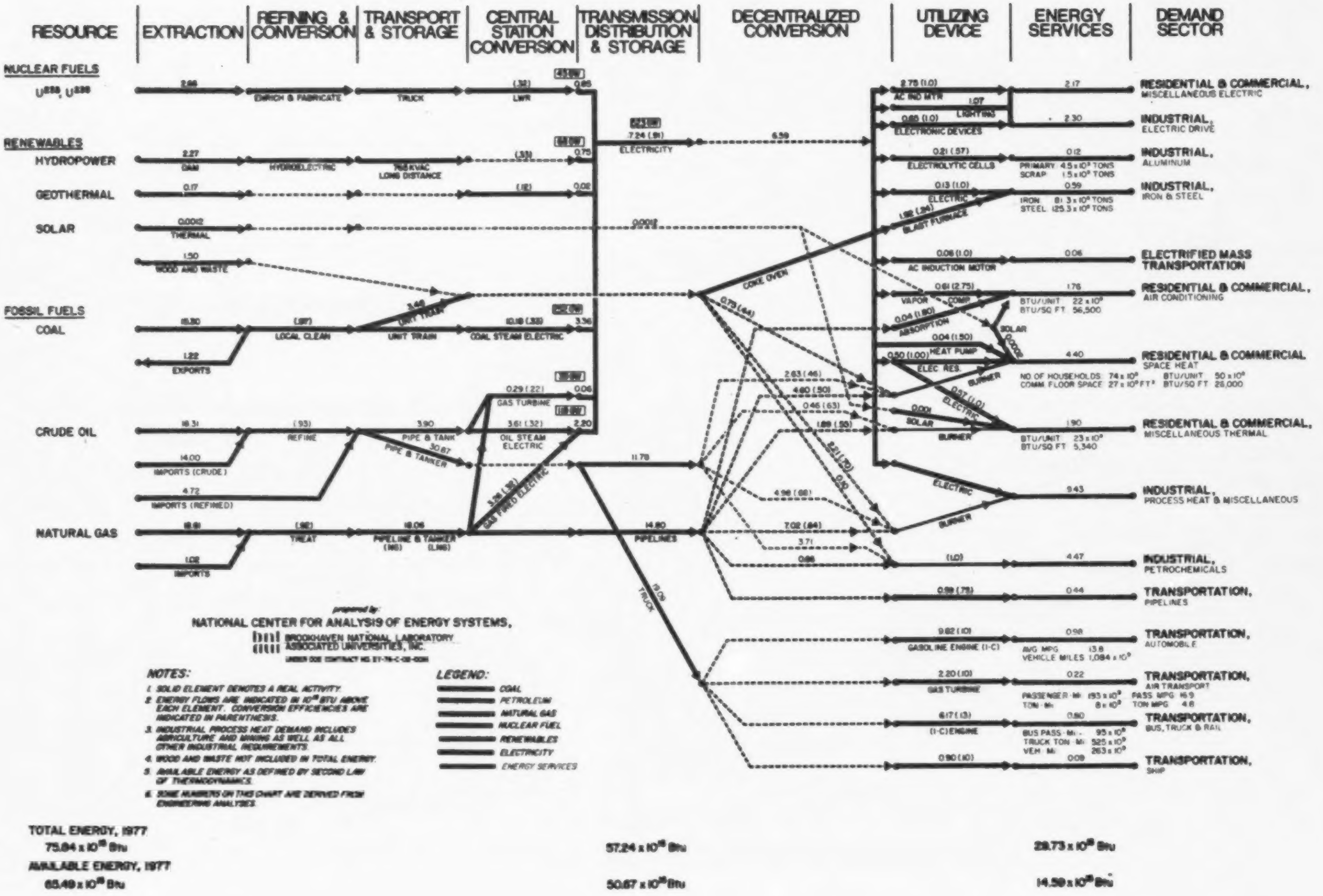


FIGURE 1

FIGURE 2 REFERENCE ENERGY SYSTEM, YEAR 1977



prepared by:
NATIONAL CENTER FOR ANALYSIS OF ENERGY SYSTEMS,
BROOKHAVEN NATIONAL LABORATORY
ASSOCIATED UNIVERSITIES, INC.
UNDER DOE CONTRACT NO. EY-76-C-02-0016

NOTES:

1. SOLID ELEMENT DENOTES A REAL ACTIVITY.
2. ENERGY FLOWS ARE INDICATED IN 10^{15} BTU ABOVE EACH ELEMENT. CONVERSION EFFICIENCIES ARE INDICATED IN PARENTHESIS.
3. INDUSTRIAL PROCESS HEAT DEMAND INCLUDES AGRICULTURE AND MINING AS WELL AS ALL OTHER INDUSTRIAL REQUIREMENTS.
4. WOOD AND WASTE NOT INCLUDED IN TOTAL ENERGY.
5. AVAILABLE ENERGY AS DEFINED BY SECOND LAW OF THERMODYNAMICS.
6. SOME NUMBERS ON THIS CHART ARE DERIVED FROM ENGINEERING ANALYSES.

FIGURE 3

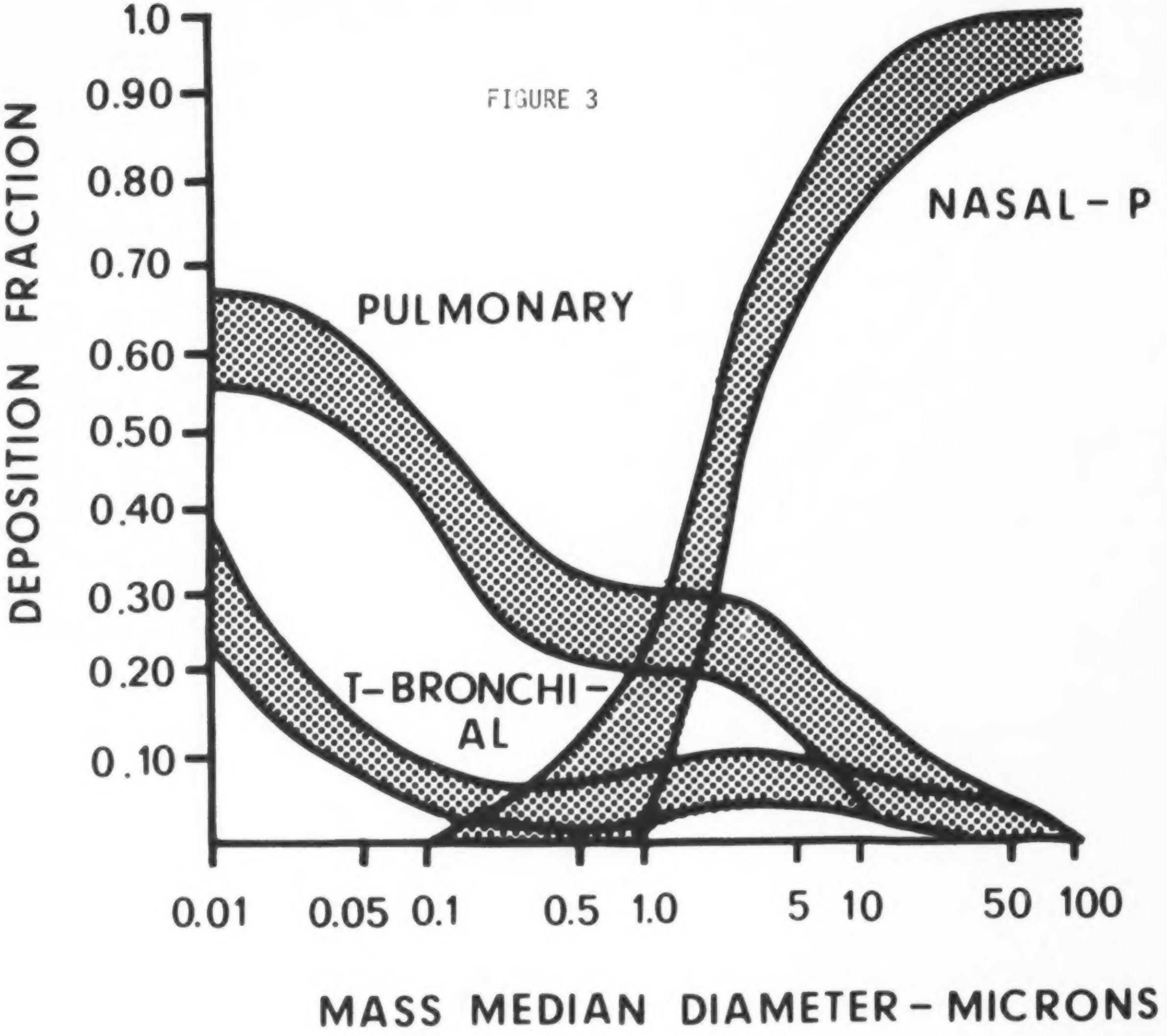

1.0
0.90
0.80
0.70
0.60
0.50
0.40
0.30
0.20
0.10
DEPOSITION FRACTION
PULMONARY
NASAL - P
T-BRONCHI-
AL
0.01
0.05
0.1
0.5
1.0
5
10
50
100
MASS MEDIAN DIAMETER - MICRONS

FIGURE 4

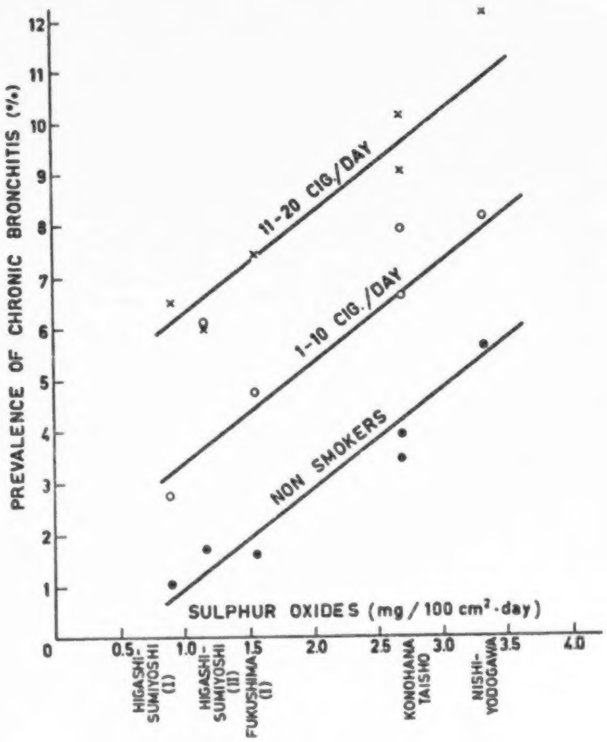


FIGURE 5

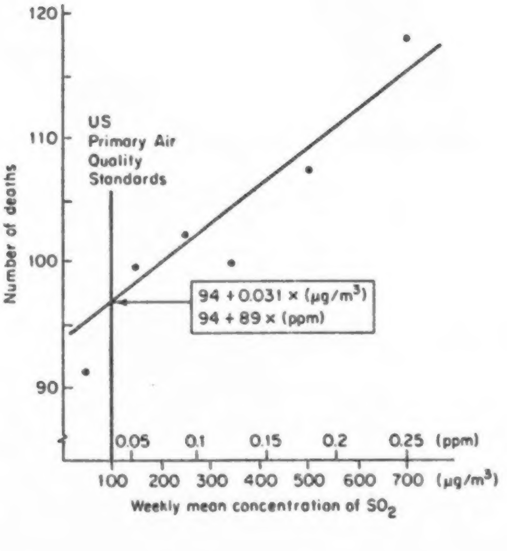


FIGURE 6

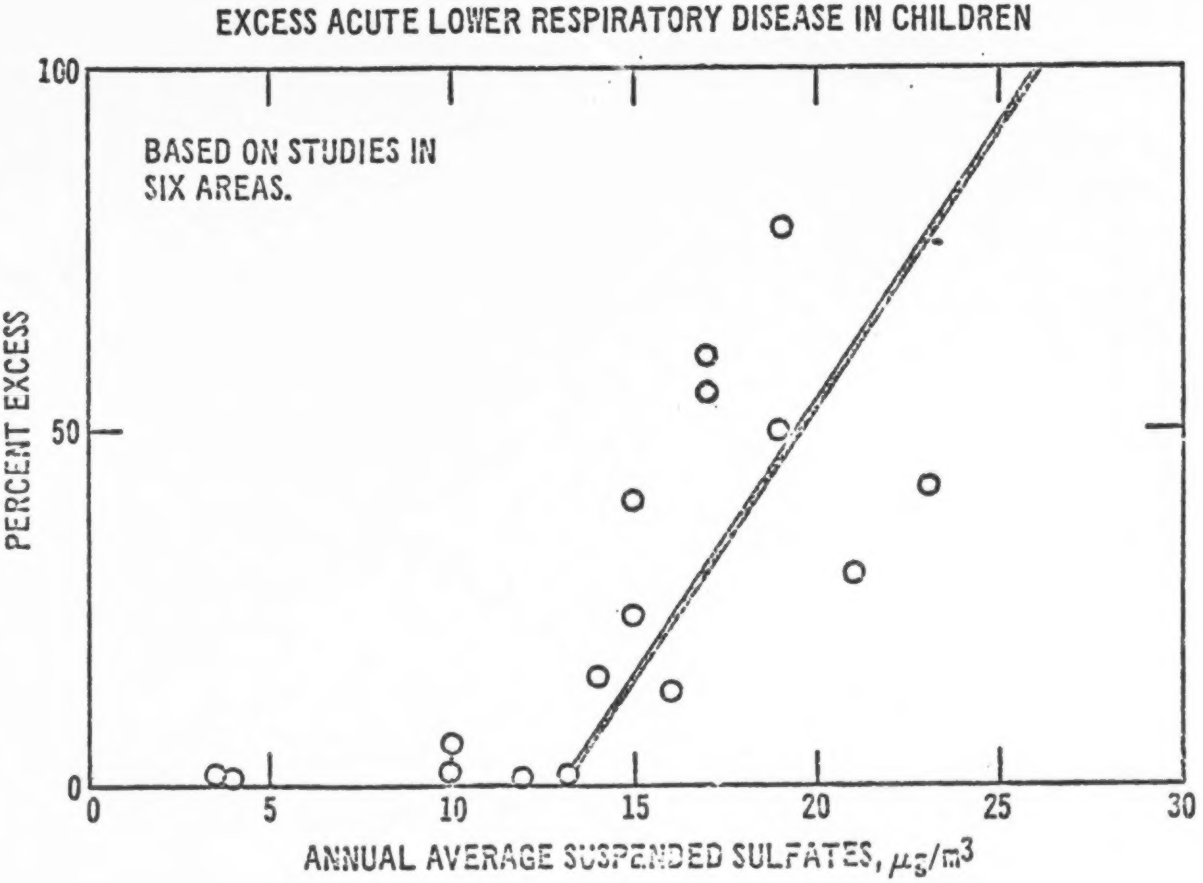

EXCESS ACUTE LOWER RESPIRATORY DISEASE IN CHILDREN
BASED ON STUDIES IN SIX AREAS.
PERCENT EXCESS
100
50
0
0
5
10
15
20
25
30
ANNUAL AVERAGE SUSPENDED SULFATES, μg/m3

FIGURE 7

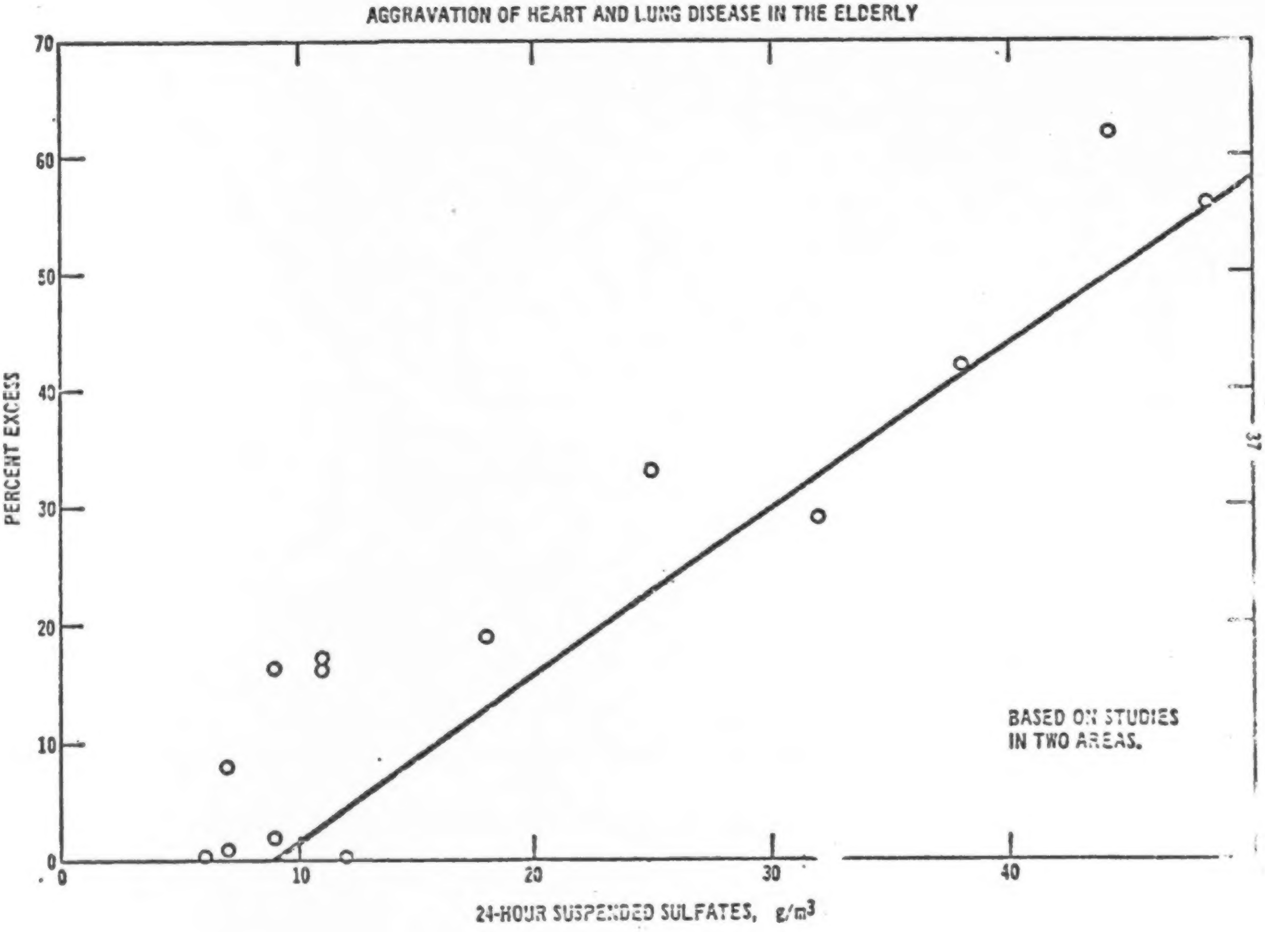

AGGRAVATION OF HEART AND LUNG DISEASE IN THE ELDERLY
PERCENT EXCESS
70
60
50
40
30
20
10
0
0
10
20
30
40
24-HOUR SUSPENDED SULFATES, g/m^3
BASED ON STUDIES IN TWO AREAS.

FIGURE 8

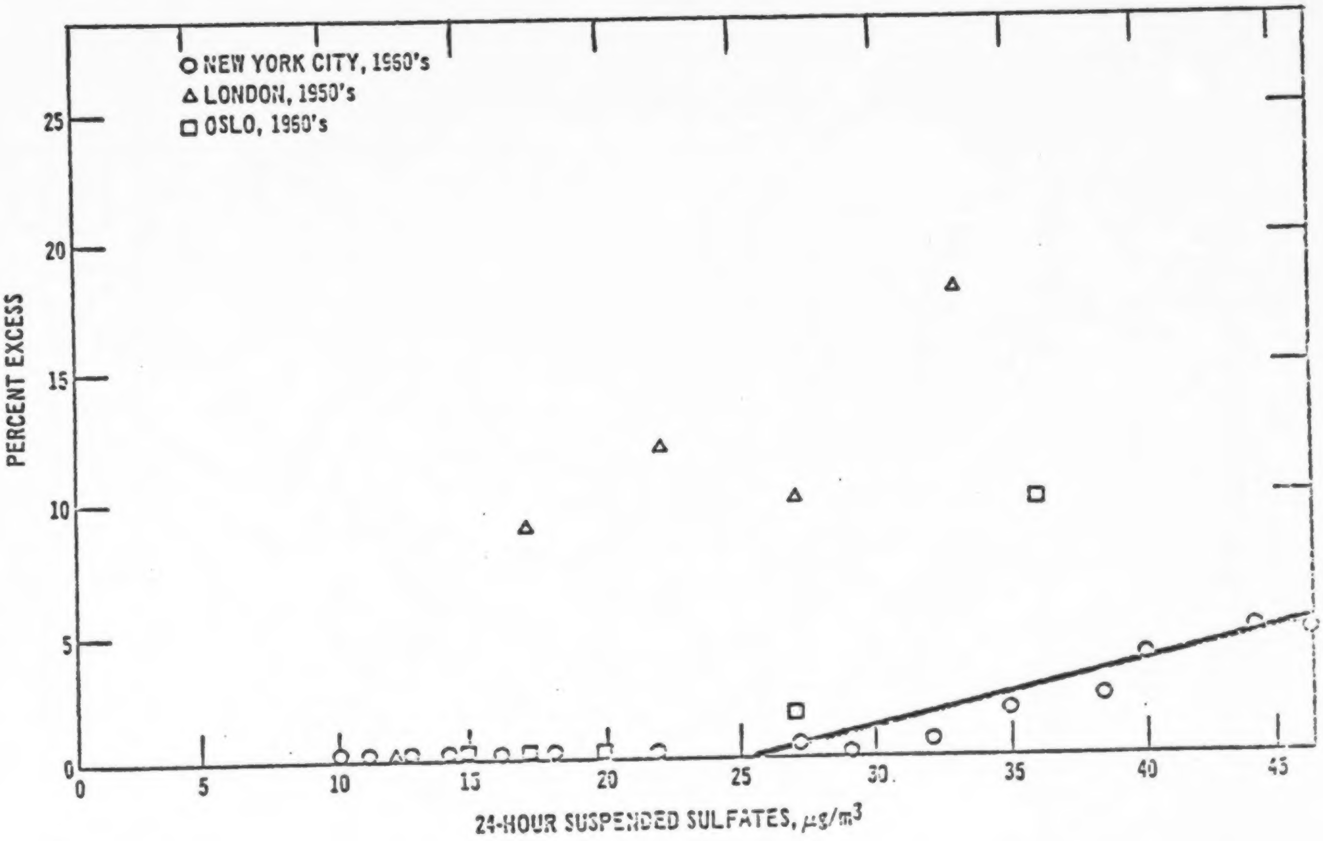
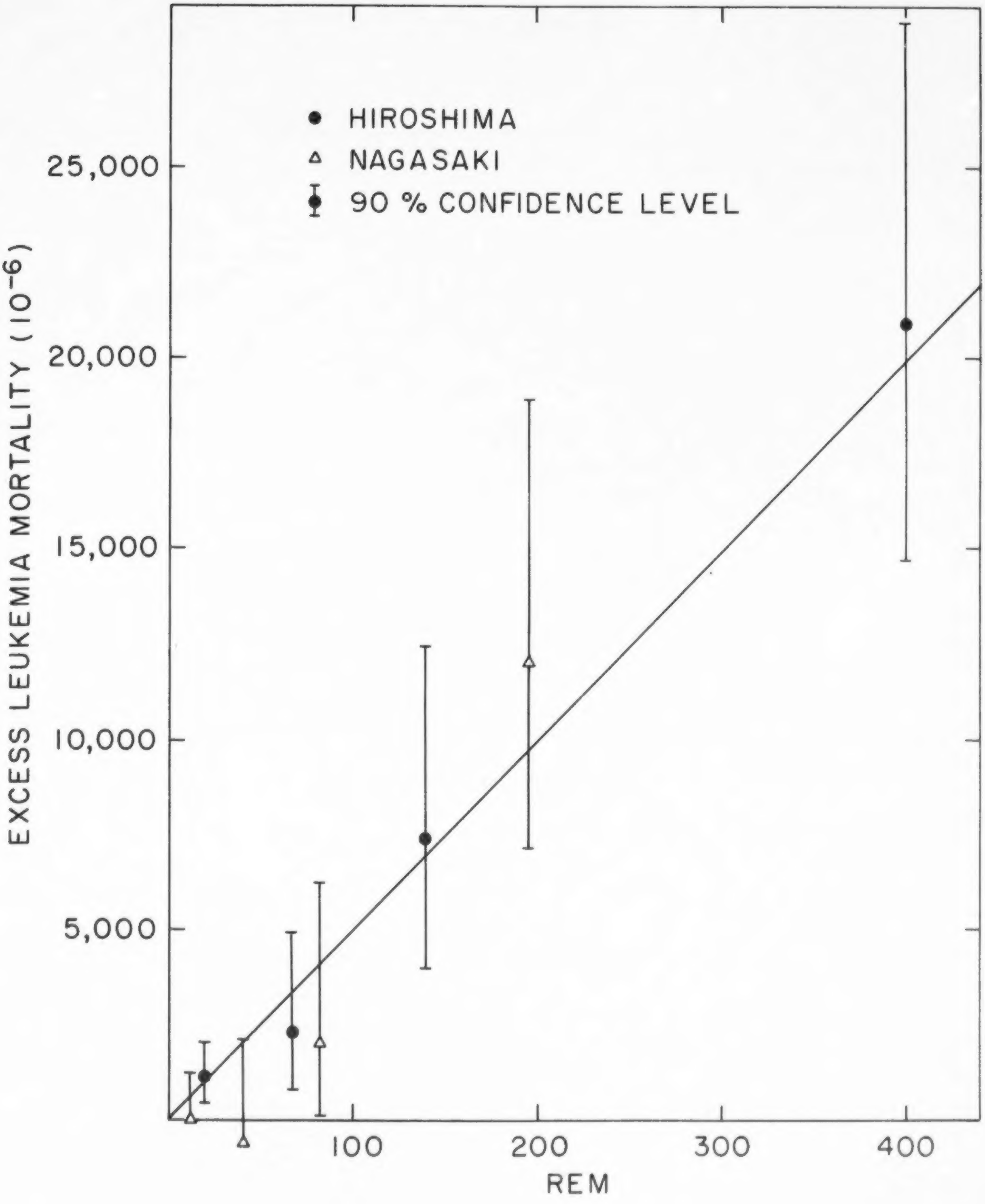

○ NEW YORK CITY, 1960's
△ LONDON, 1950's
□ OSLO, 1960's
PERCENT EXCESS
0
5
10
15
20
25
0
5
10
15
20
25
30
35
40
45
24-HOUR SUSPENDED SULFATES, $\mu g/m^3$

FIGURE 9



HIROSHIMA
NAGASAKI
90 % CONFIDENCE LEVEL
EXCESS LEUKEMIA MORTALITY (10^{-6})
25,000
20,000
15,000
10,000
5,000
100
200
300
400
REM

FIGURE 10

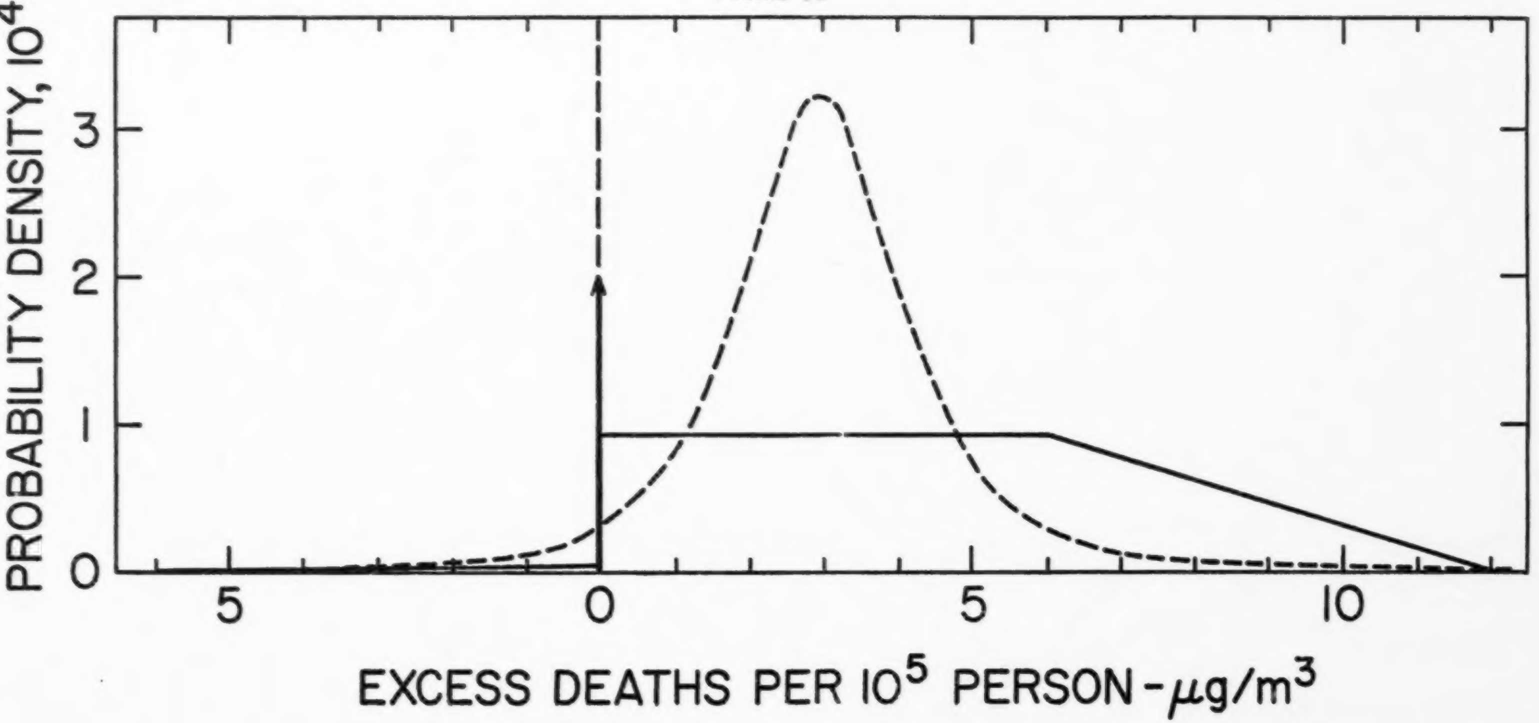

PROBABILITY DENSITY, 10^4
0
1
2
3
5
0
5
10
EXCESS DEATHS PER 10^5 PERSON-$\mu g/m^3$

Figure 11

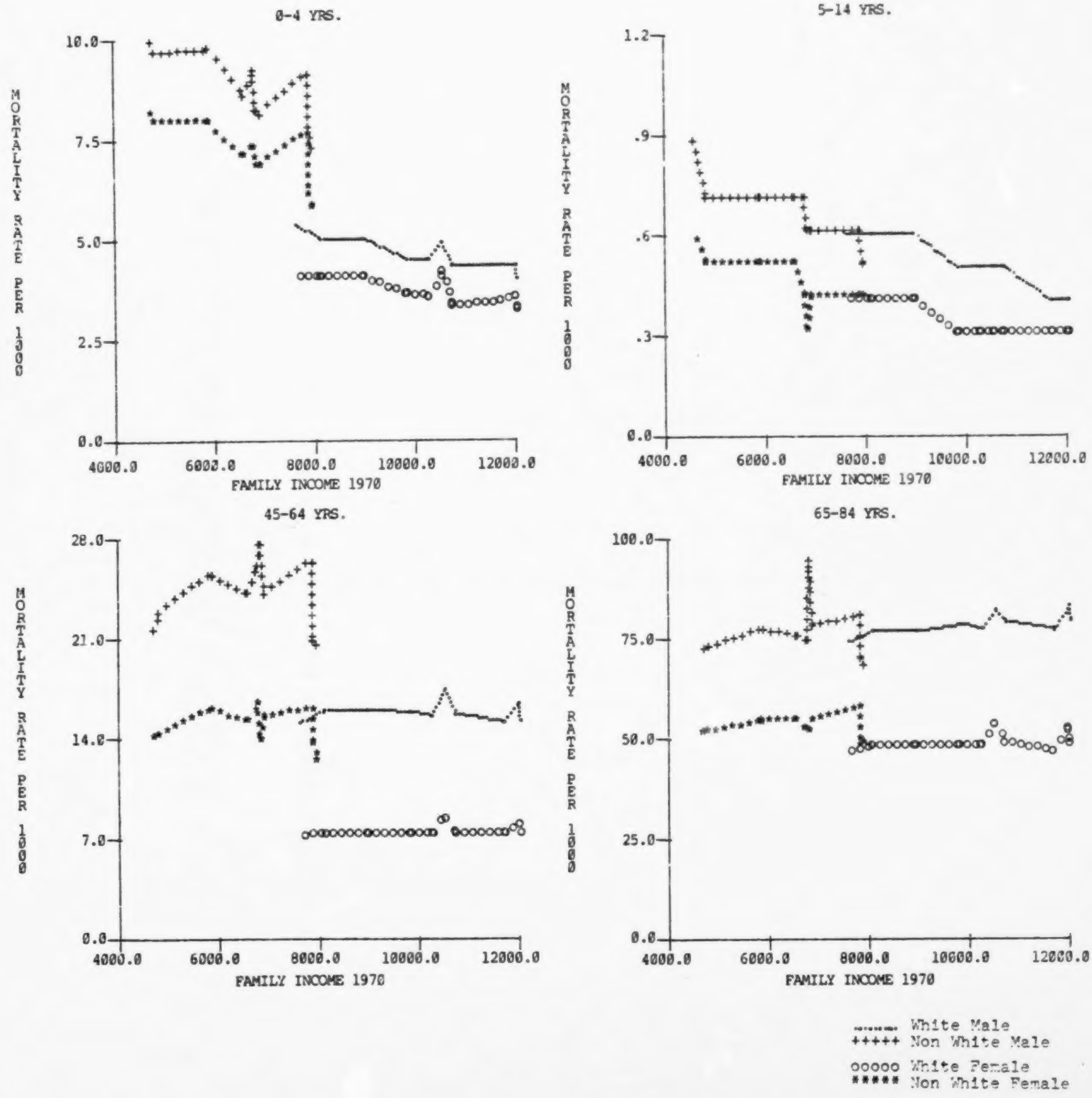
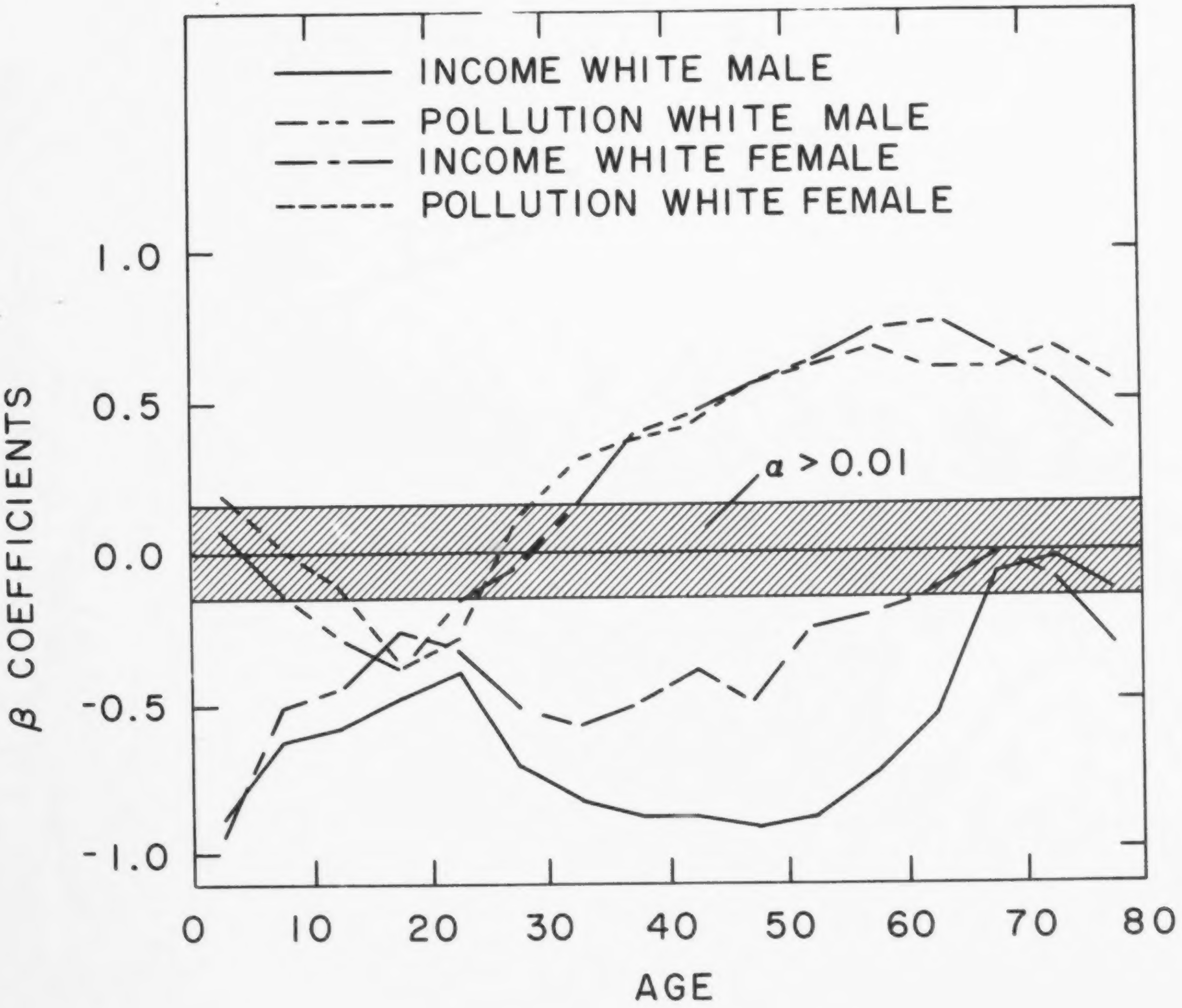

0-4 YRS.
5-14 YRS.
45-64 YRS.
65-84 YRS.
MORTALITY RATE PER 1000
FAMILY INCOME 1970
10.0
7.5
5.0
2.5
0.0
1.2
.9
.6
.3
28.0
21.0
14.0
7.0
100.0
75.0
50.0
25.0
4000.0
6000.0
8000.0
10000.0
12000.0
.... White Male
+++++ Non White Male
ooooo White Female
***** Non White Female

FIGURE 12



N.B. POINTS PLOTTED AT THE MID POINT OF EACH 5 YEAR AGE INTERVAL

FIGURE 13

Total deaths associated with SO4 exposure, U.S. 1969-71
(deaths/100,000/ug/SO4/year)

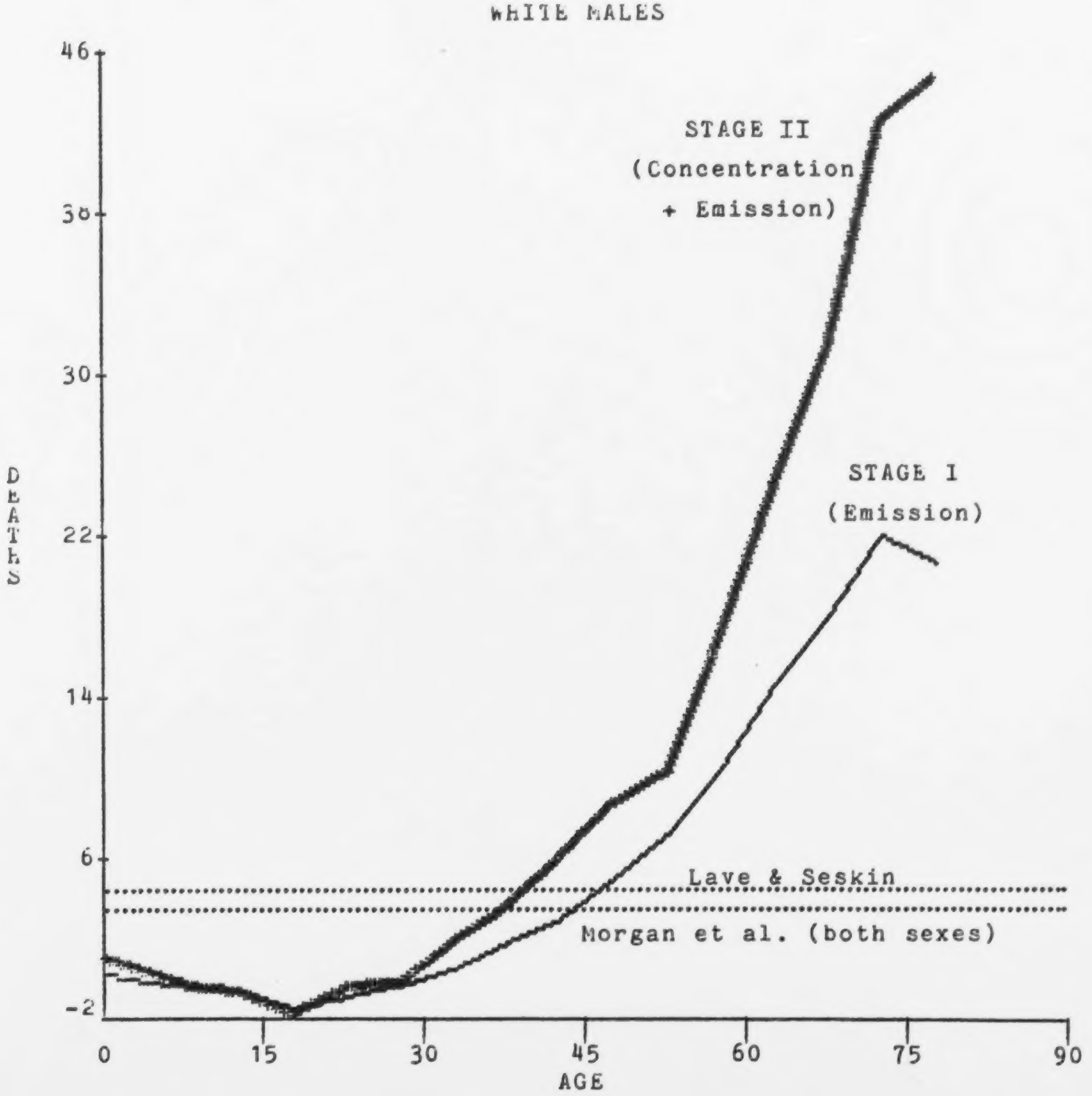


FIGURE 14

Total deaths associated with SO4 exposure, U.S. 1969-71
(deaths/100,000/ug/SO4/year)

WHITE FEMALES

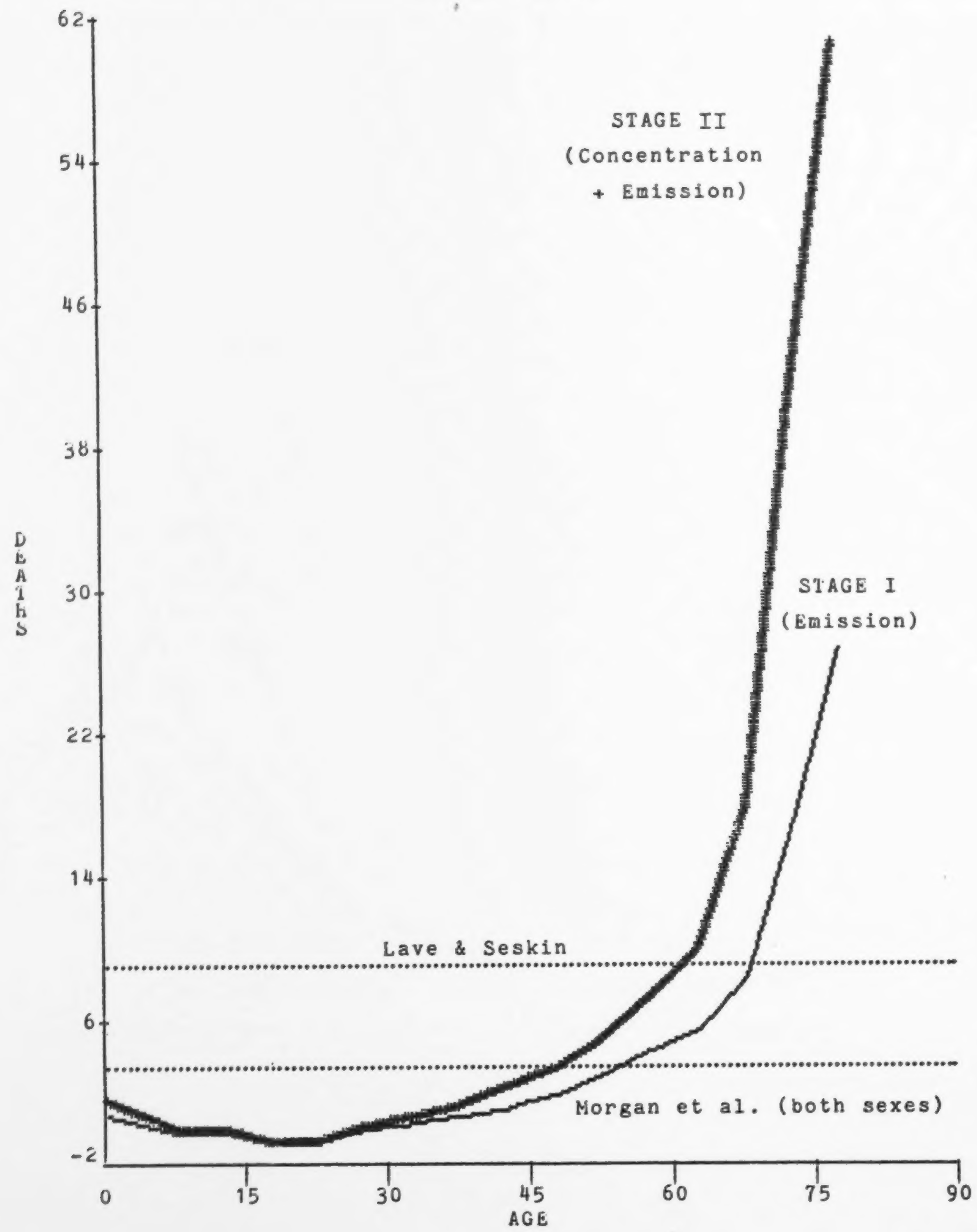
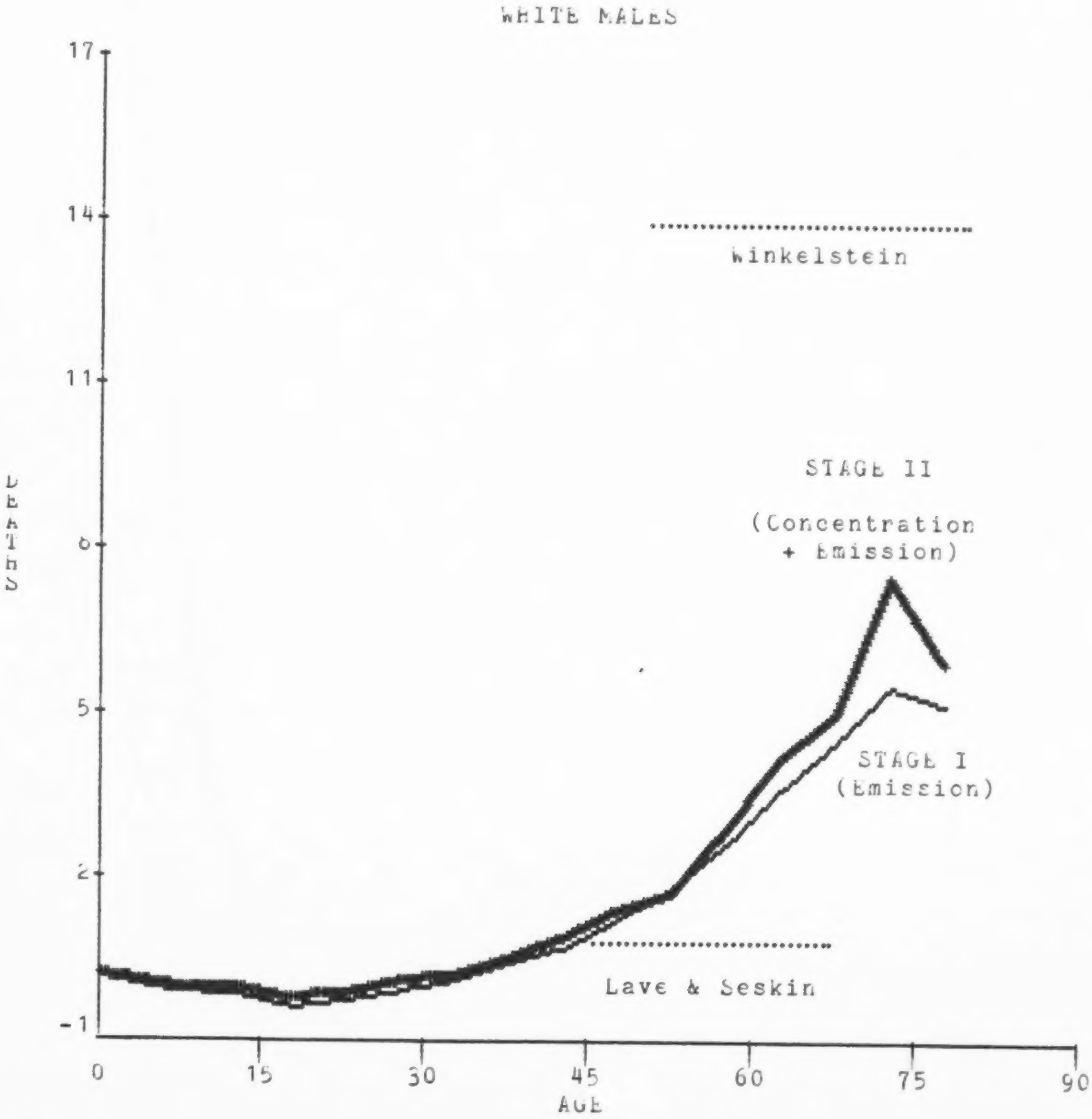


FIGURE 15

Total deaths associated with TSP exposure, U.S. 1969-71
(deaths/100,000/ug/TSP/year)



DISCUSSION

SESSION VI

Dr. Hartnett, Energy Resources Center, University of Illinois, and a member of the ORBES Core Team: This question is addressed to Dr. Hamilton. Yesterday we heard from Dr. Mazumdar, reporting on the work of a colleague, that studies in New York City showed that even though the S02 level decreased by a factor of five, there were no significant changes in deaths related to S02. Can you rationalize that with the results that you showed?

Dr. Hamilton: That's a very good question and I'm afraid I didn't make myself clear yesterday when I intervened after she spoke. I think the reason for the lack of change in New York City is due to the long-range transport of sulfate or some chemical transformation product of the S02. 80% of sulfate in New York City is foreign. Very careful study has been done on this. 80% is imported from areas of the United States west of the Hudson River, unfortunately. Again, there's another point I think you have to have some perspective on: we're looking at a very tiny proportion of the annual death rate. The worst sort of figures that we're getting from air pollution is only about 2% to 3% of the annual death rate. You'd be looking for a very tiny change on that particular point.

Dan Swartzman, University of Illinois School of Public Health: To the great relief of everybody present I have a question, not a comment. Dr. Rowe, I just wondered--you used the term "worst case assumption" in dealing with the deposition of high level radioactive materials into salt caverns. I was wondering if anybody has looked at the worst case assumption of finding out that, in fact, it doesn't work. And, in fact, we may be stuck with holding the high level wastes as we currently are, in temporary storage facilities. Has anybody done the sort of in-depth analysis that you did, which I was terrifically impressed with, for that sort of "worst case" analysis?

Dr. Rowe: Let me say, first, that I didn't do the analysis--it was done by the professional while I was heading the Office of Radiation Programs of EPA. They did look at worst case consequences. The event might be a failure of a certain kind where 50% of the total material could be released to the environment, by the worst possible pathway. This was then used as a consequence measure. An even more extreme case would involve releasing all the available material. Then they worked backwards, and tried to determine what kinds of events could cause that kind of consequences. They then began to try to base the probability of such events upon historical data and models to get some idea of their relative frequency. In a sense they have looked at some worst cases of having this material reach the environment. Now then, you ask what is the problem if we do nothing. Remember, I only stated conditions from the time the repository was closed. There are also conditions up to the time that you put the material in the repository. The major difference in concept between those is that if we do the work to put it in the repository in our generation, we are taking risks in our generation for benefits gained in our generation. But in the long term, other than the intangible benefit of the continuity of society, there is really no benefit to the future generations, so a legacy remains. If we do nothing, we leave it as a liability for the future. Once produced, wastes are nonproductive in our society. Under inflation, the worth of productive items goes up at the same time the costs go up to handle them. But for nonproductive items such as wastes, the costs escalate and become a significant part of the economy without any productivity. A good example, I think, are the 22 abandoned mill tailing piles in the West where Congress now has voted 180 million dollars to clean them up. That's a significant amount of money considering how little was spent to put them there. Another example is the culm piles in eastern Pennsylvania. These were put there at a very low cost--now we can't afford to move them and the land is not usable. The problem is further exacerbated in that the balances we make have to take into account not only the economic and the health impact aspects, but also the equity aspects.

Dan Swartzman: May I just ask one quick follow-up question because I'm not quite sure if I was understood. What I'm asking is that implicit in your analysis and much of the analyses I've seen, is the assumption that there is a method for storing high-level radioactive materials. What I'm saying is, "Has anybody done the projected health effects into the future not using that assumption?" In other words, using the assumption that, in fact, we never find a satisfying way of storing high level materials? Has anybody done the same sort of projection into the future of the health effects of that? That's what I was asking.

Dr. Rowe: I don't think such projections have been made for the total practice as I did. I think they've done it for waste we have already accumulated, a relatively a small amount, but I don't know what the effect would be for total practice.

Dr. Swartzman: Would you care to speculate?

Dr. Rowe: I only can speculate on the social system involved. We would require institutional controls to preserve the integrity of present wastes, if we leave them in the form we have now. If we did nothing, and the institutional controls broke down to some extent, we would end up with cathedrals and high priests around each repository to keep it safe. I don't know the answer.

Dr. H. Spencer, one of the ORBES Core Team members: I have worked hard in this conference to try to see to it that issues that I felt relevant and important were not overlooked. I have one more I think we passed by. It goes as follows, very briefly. The San Diego air crash, Pacific Airlines Flight 182, cockpit crew contained three pilots and one flight engineer. At the time of the crash all three pilots, one of whom was just on a jump ride, were looking for the light plane. They missed it, obviously. PSA since has flown two test flights under the exact same conditions on the same route, using a 172 separated by 1000 feet in air space for safety purposes. On both occasions, with four people in the cockpit, they could not pick out the light plane against the background which was into the sun with smoke and haze, and six miles of visibility. I want to show you people in a very quick set of slides, what six miles visibility looks like, and then I have a question to ask the group in relation to the health impact of this problem. (FIRST SLIDE) That's Madison, Indiana from an altitude of 5,500 feet, the very dead center of the American Midwest, and the visibility recorded on that day was six miles. It seems to me that from the standpoint of airline safety alone, and esthetics, this question of air pollution need not be debated or argued any further. My question is why have we not in this health analysis taken this risk to air travelers into account?

Dr. Rowe: I'll make an attempt to address the problem. If you look at the total perspective of risks in society and control of those risks, especially the involuntary ones which are imposed inequitably on people, we tend to emphasize all our efforts on those which we can do something about. Either because they're institutionally controllable or because they're technically controllable. We tend not to deal with those which are less tangible. Often for these the manner in which we do something about it requires changes in lifestyle, or changes in our whole economic structure, and in the way we live. I think this problem occurs throughout our society, not just in the particular case you cited. This is a case in which technology has fixes, and we know how to impose those fixes. We impose them through the FAA on particular air control towers in air traffic control. They are measurable and do-able. Now, I'm not suggesting that's the right approach. All I'm suggesting is that the institutions address that which they can control, as opposed to those they cannot, such as the case for sulfur dioxide.

Dr. Spencer: I want to answer that. I am an instrument-rated pilot certified for high performance aircraft and I don't agree with a thing you've said.

Dr. Hamilton: I don't want to get involved in that sort of controversy because I'm very mindful of Birch Bayh. I was very impressed by his feeling that we should try to understand what's going on, and I'm delighted at your intervention because what you've shown is, of course, exactly the distribution of sulfates in the United States. I've got a couple of slides. We don't have the time to see them but I can assure you you could superimpose "sulfates," whatever they are, on those things. And, of course, we do know that they are responsible for loss of visibility. It is a well-established fact. There have been some beautiful studies done on this, and again I think that is a very powerful argument for reducing the precursor of sulfates, which is S02. I would think, in the light of this and the long range transport, it's a powerful argument for new source performance standards, being established.

Dr. Rowe: I just have one response. Namely, I didn't say it was the correct thing to do. I happen to agree with you because I also fly a high performance aircraft myself, open cockpit as well. I happen to agree with you--I'd like to see it cleaned up. But I was just trying to give an explanation of why society doesn't seem to give equal weight to these things. That's all.

Professor Shapiro: I feel completely out of place here. I'm just between two pilots and facing one here. I am without any apology for the program, what's on here, but I would presume that if we really went into meteorology and all of the other aspects that are related to this view, we would have to have had not three days but 30 days of symposium. Hence, I would be happy if we should have another symposium on that particular subject in relation to the Ohio River Basin Study because this is of fundamental importance, but we restricted ourselves. I believe we should show more concern for accidental injury and death and agree with the need to evaluate such impacts. I happen to believe that that is also an area our school, for example, doesn't emphasize enough, and yet from the point of view of public health has tremendous impacts.

Mr. Olson, Westinghouse: I have two questions for Dr. Rowe. First, I have a comment. Dr. Rowe, you said that there is no benefit to future generations. I take issue with that because the benefit I see is that we are leaving our future generations more oil, gas, and coal by burning uranium today. My questions are both on the same slide. You showed 10,000 gigawatt years of electricity by burning uranium. Is that with or without plutonium recycle? Secondly, then you showed only 10 times as much for light water reactors. Why shouldn't that be more like 60 times as much?

Dr. Rowe: The 10,000 gigawatt years are for the burning of uranium in our present fuel cycle operation. With plutonium recycle and thorium, I extended that by a factor of three. With the use of breeder reactors, I extended that by a factor of 100. The idea being that with the breeder reactor we may or may not get the breeding ratios we are talking about; so assuming one order of magnitude of uncertainty here is not going to affect the five orders of magnitude uncertainty that I was talking about at the endpoint. Regarding your first question, I think you misunderstood me. I didn't say there was no value. I said there was a value to the continuity of society with or without gas and oil.

Ed Light, Appalachian Research and Defense Fund: I have a couple of questions for Dr. Hamilton. First of all, if ORBES is able to come up with some estimated sulfate concentrations for projected power plant emission scenarios for the next 20 or 30 years, and have estimates as far as the populations residing in different areas of different sulfate concentrations, could they use your estimates of sulfate mortality effects to come up with some projected mortality in this area?

Dr. Hamilton: Yes, very definitely.

Ed Light: The other question is: Could you suggest to us how we could estimate the morbidity factors of having these various sulfate levels in the different areas around the power plants?

Dr. Hamilton: Yes, I'm very glad you've asked the morbidity question because I didn't touch on that. I think that you can use several methods. First of all, we do have a rough factor by which we multiply mortality. If people are going to die from air pollution, our view is that it is not a harvesting effect, our view is that it is a chronic effect and we do have some estimates on person-years lost. The other point of view is that one should try to use some of the best EPA estimates, guesses, for shorter periods for acute effects. They don't have such things for chronic effects but they have them calculated both for TSP and for sulfates and, with all the caveats that one wishes to include in them, I believe OTA has suggested that they're reasonable to use. I feel we're going to start trying to use them, examining the uncertainties involved with them very clearly.

Prof. Shapiro: I'd like to ask Dr. Hamilton to address the problem that can be seen in the studies that he referred to and studies in other areas of health effects which exhibit a similar kind of problem. Namely, that when we conduct the analysis on a global basis, as when we evaluate relationships between "sulfates" and the whole population of the United States, or a very large population, you obtain these relationships which indicate a health damage due to "sulfates." But when you start analyzing smaller aggregates, those kinds of effects seem to vanish or at least are reduced. What is the reason for such findings?

Dr. Hamilton: You're not referring now to the actual use of the damage function; you are referring to its derivation. The question is a very interesting question that Professor Shapiro has raised, and it is one that was demonstrated very sharply by Lipfert's reanalysis of Lave and Seskin's work using the same air pollution monitoring stations, and the data from that for the inner city portion of the standard Metropolitan Statistical Areas. Lipfert demonstrated that when you restrict the mortality to consideration of only people in the central city, this apparent correlation between "sulfate" and mortality, this association, disappeared. You want my explanation for that? My explanation is very straightforward and simple, as far as Lave and Seskin's work and its reanalysis. It is that when you restrict yourself to the inner city, you have very powerful-- particularly for the period that we're dealing with-- socio-economic impacts of the inner-city poor. That was a period, by the way, when there was tremendous non-white in-migration into those areas. I have already shown you that the economic variable, as a matter of fact, is a very powerful variable. I believe that is why it disappears. It is interesting that the association between total suspended particulates and mortality does not disappear in the inner city. I am not able to explain that differential thing. This Lave and Seskin's type of analysis, which is a multi-regression analysis, is very sensitive to the weight of the different variables. If you have a very powerful variable, which obviously comes out when you go to the inner-city, I think that explains why that disappeared. That objection, by the way, doesn't apply to the work of Bozzo, Galdos, Novak and Hamilton, which covers the entire country.

Professor Shapiro: But when you use the Standard Metropolitan Area as an area of study, you're including a tremendously variable set of circumstances, both from meteorological and from population density points of view. Where you locate your measuring devices seems to me very important. So I can't see how you can, on one hand, have it disappear and explain it on an economic basis, but then it appears when you use this tremendous area. It just somehow doesn't make sense to me.

Dr. Hamilton: Well, you made a very good point of uncertainty here because we are using a single station; but we're increasing the sensitivity of the analysis to pollution because we are using a larger area, a larger mortality base, and we're damping out the effect of poverty. That would be the explanation. Let me just say you've put your finger on a very important area of uncertainty. The use, for example, in our analysis of only 248 stations for the entire 3,100 counties, even though we've gone through a very deliberate aggregation analysis based on our previous work on emissions for all the counties, is not a desirable thing. We already know that there is great uncertainty anyway from one single air pollution monitor in an area and people's outdoor and indoor exposure. But, you know, that's the best analysis we can come up with at the present day. Until we have studies where you have personal monitors attached on people, and follow them for the next 20 or 30 years, we're not going to have any better analysis. That's my point. You've got to have some assessment.

Dr. Radford: Well, it seems to me that to rely for damage functions on a very blunt tool, which the multiple regression analysis is, and which, as you know, is fraught with a great deal of controversy and question as to whether, in fact, it shows any correlation or not, and to ignore what I would call much more specific studies, in which an attempt has been made to directly relate exposure to sulfates, sulfur oxides, and so forth, in areas in relatively small populations where you can have single exposures, I think that is scientifically not very sound. The impression I have been left with, especially by data from multiple regression analysis, the damage functions are just not known. To assume that we can regulate sulfur emissions on the basis of health effects when there is that much uncertainty, I think from a regulatory standpoint, would be a disaster because it would be shot down in the courts in nothing flat. A point that Dr. Lippmann made yesterday was really reinforced by what you just said. We may be barking up the wrong tree. What we should be concerned about are some of these broader effects, the impact of sulfur oxides combined with ozone on plant life, as well as things like visibility, etc. These may be where the action is in terms of controlling power plant pollutants from coal burning. I would just like to leave it on that note and maybe get a reaction from the panel, as to whether we can at this stage in our regulatory process, do anything on the basis of other than putative health effects, which a lot of us are very uneasy about.

Dr. Hamilton: Well, I'd like to answer Dr. Radford in this area. There are an overwhelming mass of independent studies, independent of these cross-sectional city studies, the Lave and Seskin work, and our own massive work in this area. They point undeniably to the fact that some transformation products of SO2 in urban atmosphere is harmful to health. Now, the fact that Dr. Ferris hasn't seen it in his small sample in the Six-City Studies, he has not been able to pick up anything yet so far, doesn't negate the fact that air pollution is bad for you. There have been all these episodes. It raises some questions about dose/effect relationships. But he hasn't given us any figures for what would be the lower limit of risk, as a matter of fact, that you might be able to derive from his studies. So, it seems to me that if you need some quantitative measure of damage, and I think one does in any assessment, and we're not talking about instituting sulfate standards or any other, suppress the precursor of this material, which is the SO2. And I don't think anybody denies that. Dr. Ferris himself said that he wanted to live with the present standards, apparently. I don't know what that meant, mind you, since I don't know whether he meant he was against new source performance standards or what? He seemed to avoid the issue as to whether or not sulfates or some material, related to the oxidation of SO2, is harmful to human health. I don't know. I'd like to hear from somebody else on this.

Dr. Andelman, Graduate School of Public Health, University of Pittsburgh: I'd like to ask a question as a non-pilot, as a highly rated rower of boats, and a peruser of water. In the water field today, there is a considerable problem in setting criteria. EPA is going through this exercise right now for their 129 priority pollutants, many of which, if not the greatest majority, are, in fact, of concern from a chronic health point of view. Now, much of the discussion, although not all, in relation to air pollution for the past three days, has been focused on such things as sulfur dioxide, nitrogen oxides, and particulates, presumably based on their more acute effect concerns. I'd like to ask perhaps Dr. Hamilton or the others, what might be the area of concern on air pollution exposures for these many emissions of varying kinds of pollutants from power generation, about which we probably similarly have very little good information regarding their health effects; and might these ultimately prove to be of such considerable concern that even when we can control some of the more acute kinds of pollutants, these others might still end up being very massive in terms of their possible impact.

Dr. Hamilton: I assume you are referring to the non-criteria pollutants, such as polycyclic aromatic hydrocarbons and trace metals, etc. Well, let me put it this way. The strategy we have been advocating in dealing with this thing, is that if you reduce particulates and SO2, you will also reduce the trace metals and polycyclics that are going to come out too (and also nitrogen oxides). As long as you do a thorough job in minimizing those things, you should pari passu reduce these other possible potential bad actors. All the modeling studies that have been done on trace metals, in association with burning larger amounts of coal from fossil fuel power plants, and for that matter, from the polycyclic aromatics, have indicated that we don't anticipate that there would be large amounts.

Dr. Rowe: Dr. Hamilton used the word "strategy" in what he was talking about. I think that'a a good opportunity to separate the technical information knowledge and certainties we have from what I would call the social or political ones. You brought up the problem of water pollution. It seems to me in the area of water pollution we spend more money than any place else in our economy for pollution control, and very little of it is for health effects--except for the drinking water problem and perhaps some particular contaminated areas. Most of it is for maintaining or reclaiming our resources. I'm not indicating whether this expenditure is good or bad, but a tremendous deal of money is spent in this area for municipal treatment plants or maintenance of existing resources. The problem is, we do have limited amounts of resources that we can put into taking care of pollution. I think it is possible to begin to take our resource estimates and to look at our technical uncertainties and to determine, based upon the range of our uncertainties and the cost of control at different levels, where we can best spend our resources; even to make judgments about the uncertainties. The problem is that at the present time all of our various pollution laws are set up under different acts and we're constrained by the institutional constraints. We haven't looked across our society to find out how to best allocate our resources. We can possibly begin with the technical uncertainties. It's not just the problem here for energy development by itself; I think it's across the board, a measure of how we address society in a cost-effective manner.

Professor Shapiro: I'd like to take, if I understood you correctly, slight issue with you and point out that in this area of synthetic fuel production, or coal conversion to liquid fuels and gases, where we know that it produces damaging material, and therefore we start not with the assumption of the cost of how much it will cost to control, but that if we don't institute truly preventive measures, then we'll be way off beam when it goes on stream. So I think there are areas, especially where we start de novo, of a need of instituting the best, and even in a sense an over-control, rather than an under-control.

Dr. Rowe: I'm not necessarily in disagreement with you, except I recognize the fact that it's much more effective to be able to regulate at the beginning than it is to try to retrofit. Even for that reason alone I might agree with you. But as a second aspect, I think some of the practices that we've accepted in the past have been perhaps lax in terms of pollution control. Any new process coming in, no matter what it is, is now subject to a different scrutiny; and no matter what we do, I suspect our society will be more conservative. For right or for wrong, I think it's a fact of life.

Ed Light: I would like to bring up an issue which I haven't heard addressed yet, but since one of the topics on the program this afternoon is remaining areas of uncertainty, I think it is very relevant. That is the question of the risk of an accident from a nuclear power plant. I think this is a very important issue for ORBES to address in terms of what are the health effects and what is the probability of this occurring. One of the primary reasons for ORBES being formed, and one of the primary uses of the information which will be generated by ORBES, is to help decide how much we want to go towards nuclear and how much we want to go towards coal burning power plants in the region. The expert opinion we have received so far in this conference, are suggestions that if all segments in the fuel cycle go according to standards, there really aren't very significant health problems. I mention here that I am somewhat leery because we've gotten mainly one side of the expert view on this issue, and perhaps we have excluded a body of opinion from people with maybe an equal stature as experts, which may have different ways of looking at some of the same data. But if this is true that the well-run nuclear plants are not much of a health risk, what is the best information we can develop in terms of the risk of some type of accident developing where things don't go according to standards and we have a big release of radiation from the plants?

Dr. Hamilton: I'm very glad you have asked that question. I want to apologize. The shortage of time prevented me from following the example of my colleague from Brookhaven, Dr. Morris, and showing you a film clip from the China Syndrome; but I understand the film is on locally, and I recommend that as the first move in answer to this question. But now, seriously, I was involved, my group was involved, and we had Dr. Wald who was also involved, in the revision of the consequences of the reactor safety report. I actually played a very interesting intermediary role between the reactor safety report group and the American Physical Society group that was criticizing the reactor safety report. I think we arrived, as far as the biological consequences were concerned, at some reasonable meeting of the minds and very fortunately we have on the panel, today, Dr. Bill Rowe, because he was the chap who wrote the EPA criticism of that part of the report. In any case, we now have to live with the fact that the report has been reviewed by yet another group, the Lewis Committee, and essentially what the Lewis Committee said is that, on the whole, the methodology was fine, but they felt the limits of uncertainty were greater. I mean that is the key issue that they raised. They also attacked the use of these probabilities when you were very uncertain, when you really didn't know whether there was any truth in it or not, and I think that was a very serious criticism. Now, with that point, I'd like to turn it over.

Dr. Rowe: Let me just say I was a member of the Lewis Committee also. You stated your position very clearly. The problem that I wanted to address in my talk is one problem, that of high level waste for a nuclear situation. There are lots of problems in nuclear energy. There are lots of problems in coal and oil as well. The problems may not always be commensurate on the same scale. On the other hand, for the nuclear case, I brought up a different problem: namely, that of the problem of disposing of uranium mill tailings. I think that's an area that hasn't yet been solved. In the area of the accident situation, the Lewis Committee did state that the uncertainties were greater than were stated in WASH-1400. But they could be either way; they could be lower as well as higher. I think we have to do some more work in that area.

In the case of oil, for example, I think this is where we have our highest death rate per whatever energy measure you want to use. This occurs in the occupational area, because of loss of tankers at sea, especially those under flags of convenience, where the counting of the number of people versus the use of oil, has never really been statistically added up in useful ways.

Dr. Spencer: Once again I want to go along with what Dr. Hamilton said earlier. We've looked at some of the sulfate data, and up through 1977 the smear of haze does superimpose over the sulfate distribution. That's not my reason for coming here at the moment. Let's suppose that we don't have the Mancuso, Stewart, Nealy Report, or the study of the ship-yard workers, or the problem of the children in the desert subjected to fallout from the bomb test. Do you feel, Dr. Hamilton, based on our state of knowledge, that the regulations are weak, strong, or somewhere in between? What is your professional opinion on that question?

Dr. Hamilton: Well, my reaction was that I have always felt rather uncomfortable with this business of dividing the year into quarters and allowing people somehow to get 12 rems a year. I only discovered that relatively recently in my life, as a matter of fact. I feel that somehow or other we should have a 5 rem a year standard. I like the principle of as low as reasonably achievable, whatever it is. But I like even more what I've learned about the naval ship yards activities insofar as they have had a forcing operation and I've seen them reduce the level down to, I think the figure sticks in my mind, about 2 rem a year per person. I mean, that's my off-the-cuff reaction to that question.

Dr. Posvar: May I suggest, since we're losing a lot of our audience, and I believe our speakers would be willing to stay for further informal discussion, that we we limit ourselves to one or two more questions in this formal part of the presentation.

John Campbell: There was a little talk about the Rasmussen Report and I just wanted to ask and see if I can get my impression of it confirmed. Is it true that the Rasmussen Report is still just about the most reliable accident safety study available at this time?

Dr. Hamilton: Well, it is the most because it's the only one. It's also the least; whichever way you want to look at it.

John Campbell: Okay, now on the upper bounds of the uncertainty; can you, on the basis of reactor operation to date, set an upper bound on the probability of an accident?

Dr. Rowe: There are two ways of looking at this problem and the first is to recognize that there are different kinds of reactors operating under different conditions. The question is, can you lump them all together, including naval reactors, for example, and use that as an upper limit? I don't know if you can or not. I don't think the differences have been looked into in far enough depth yet to know if this is feasible. I could take, perhaps, a group of reactors, operating under similar conditions, and be able to set upper limits for that particular set of reactors with some confidence--but to lump them all together, I don't know yet if this is proper or improper. Secondly, there is something else going on which we have yet to measure. This is the whole operation imbedded in a regulatory process. When something occurs, a fix is made. This fix may even lower the probability for the very low probable accidents. So we don't know yet what the effects of an ongoing regulatory process are on safety. Until we know that, we may find that even our history of data bases is getting better--so it could go either way. My answer would be that, in terms of upper limits, we can take the history of collected data we presently have and lump it together and get some figure. Whether that's a good estimate of what is really going on or not is another question. I think it is premature to say.

Dr. Hamilton: I'd like to tell a little story about the use of the word, "both." The use of the word, "both", I found since I came to this country, is grossly abused by many people; it's real academese. The only correct use is illustrated by a marvelous movie in which Humphrey Bogart played, called "Beat the Devil." Humphrey was asked by a North African Potentate which he thought was more chic, a Rolls Royce or a Cadillac. Bogart said, "you should have both." That epitomizes my attitude toward this question of nuclear and coal, by the way, that I wanted to get into the record. From the health point of view, I think you can have either. I see nothing preventing control of both of them equally well. It seems to me on the health point of view, I would like to leave it on the record that I see nothing to choose with them. It's possible with proper controls for both to reduce the health effects to really trivial proportions.

Dr. Posvar: I think that's a good moderate tone on which to end this discussion. I want to thank Professor Shapiro, Dr. Rowe and Dr. Hamilton for their excellent presentations. I'm sure you will all join me in this sentiment. As a layman, I must say I haven't heard much here this afternoon to give me good cheer, but I am very much impressed by your concerns and by the expertise of the people in this group. Now I would like to call on Dr. Radford to close the meeting.

Dr. Radford: Thank you, Chancellor Posvar. I will put it in the record, and it is really relevant to a point that Ed Light asked. I don't think everything is peaches and cream in the nuclear fuel cycle anymore than I think it's peaches and cream in the coal or oil or any of the others. I think we have a lot of problem areas. One of the things that I sense, at least speaking personally, is that I can now sharpen my focus on certain areas. For example, specifically, the occupational hazards in the nuclear fuel cycle. I think they are relatively out of control, notwithstanding what Bob Minogue said today. I think they have exemplified the fact that the sort of sense, "well, everything is O.K., so we can go on doing things the way we have been doing" has been worn out by history; that a number of the reactors, as well as perhaps even some of the nuclear facilities other than reactors, are not achieving what I consider to be a safe level for the workers, while some of the others are. Once you find out that some are, you ask the question why aren't all of them? On that note, I think it is appropriate to perhaps close the formal portion of the session. I want to thank all of the audience and all of the speakers and moderators personally for their good work, and I personally am looking forward to a review of this transcript because we do want to incorporate all of this material in the final report, and it will influence the ORBES Report when it finally comes out. Thank you all.

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